# City of Portland Bureau of Water Works

## Models for Lead Exposure through Portland's Drinking Water Technical Memorandum 1- Final Report

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## Introduction and Purpose

The City of Portland is the largest supplier of drinking water in Oregon. The Bureau of Water Works (Water Bureau) provides water to about 500,000 people within its service area, and to an additional 300,000 people in the Portland metropolitan area through wholesale water sales to 19 public water systems.

In 1991, the EPA promulgated the Lead and Copper Rule (LCR) to reduce lead and copper in water at customer taps. LCR requirements include "optimal" corrosion control treatment to minimize lead and copper at the customer's tap. For Portland, such treatment would involve increasing the pH of Bull Run water from current levels of about 6.8 to 9.0-9.5, and increasing alkalinity from current levels of 6-12 mg/L to at least 25 mg/L as $CaCO_3$.

In June 1994, the Portland City Council directed the Water Bureau to investigate alternatives for LCR compliance. Part of that investigation included a study to determine the effects of corrosion control treatment on lead exposure through Portland's drinking water, and to determine what, if any, health benefits may result to the community from treatment. The purpose of this report is to present the results of that study.

## Models for Health Effects of Lead Exposure

Lead in the environment is associated with a variety of adverse health effects. Lead is most hazardous to children under the age of six, whose still developing nervous systems are particularly vulnerable to lead and whose normal activities expose them to lead-contaminated dust and soil. The Centers for Disease Control currently indicates that the lowest blood lead level of concern is 10 ug/dL. Some studies have suggested harmful effects at even lower levels, but the body of information accumulated so far is not adequate for effects below about 10 ug/dL to be evaluated definitively.

Two types of health effects models were developed as part of this study. The "individual-based" model was used to estimate the contribution from lead in water to the blood lead level of an individual infant, child, or adult before and after implementation of various levels of corrosion control treatment. The "population-based" model was used to compare the distribution of blood lead levels before corrosion control treatment for infant, children and adult populations in Portland to predicted distributions after implementation of various levels of corrosion control treatment.

Three corrosion control treatment alternatives were selected for modeling that involve pH adjustment to the following ranges:

| Level of Corrosion Control Treatment | pH in distribution system | Predicted reduction in water lead levels as compared with baseline levels |
|---|---|---|
| Baseline: no corrosion control treatment | <= 7.0 | N/A |
| Alternative 1: Limited corrosion control treatment | 7.0-7.5 | 40% |
| Alternative 2: Moderate corrosion control treatment | 8.0-8.5 | 60% |
| Alternative 3: LCR-defined "optimal" treatment to minimize lead and copper | 9.0-9.5 | 70% |

The models require various types of input data, including the distribution of water lead levels and blood lead levels in Portland before corrosion control treatment, the anticipated reduction of water lead levels with corrosion control treatment, and coefficients relating the contribution of water lead to blood lead levels. These models make use of best available input data. Results are not intended to be interpreted on a precise quantitative basis, but rather should be used as relative indicators of potential reductions in blood lead levels under various lead exposure scenarios.

**Findings**

- The sources of lead in Portland's drinking water are located in premise plumbing.

  There is no detectable amount of lead in Portland's source water. The main sources of lead in Portland's tap water are lead-based solder that was used before 1985 to join copper pipes, and brass fixtures that can contain up to 8% lead. Lead may be "picked up" from these sources if water stands motionless in premise plumbing systems for many hours. Running water typically has little or no detectable lead.

- The prevalence of elevated blood lead levels in Portland is low.

  The CDC (1995) proposed a definition of low-prevalence communities as those in which 13% or less of children have blood lead levels >= 10 ug/dL. Because only an estimated 6% of children in Portland have such levels, Portland would be considered a low-prevalence community under this definition.

- Lead-based paint is the most commonly identified source of elevated blood lead levels in Multnomah County.

  The Multnomah County Health Department has conducted over 120 follow-up investigations of EBLLs in Multnomah County and the Portland metropolitan area since 1993. Analysis of the results of these investigations indicates that lead-based paint is the most likely source of exposure for 70% of the EBLLs of at least 15 ug/dL, and for 80% of the EBLLs of at least 20 ug/dL. These observations are consistent with the CDC's statement that lead-based paint is the most common high-dose source of lead exposure for children.

- Water is not a major route of lead exposure in Portland.

  Based on analysis of best available data, the median lead level in running water samples is estimated at < 1 ug/L (below the detection limit). The FDA has set a limit of 5 ug/L limit on lead in bottled water. It is estimated that about 95% of the running tap water samples and about 70% of the standing tap water samples in Portland meet the FDA standard.

  However, for a set of unlikely exposure conditions; it is possible that water could provide a significant contribution to an individual's blood lead level. In order for water to significantly contribute to an individual's blood lead level, that individual's water consumption would likely have to consist solely of standing water with elevated lead levels.

  For example, consider the highly unlikely exposure scenario of an individual infant consistently consuming standing water with 49 ug/L of lead (the 90th percentile standing lead level in Portland's Tier 1 homes in 1992). A blood lead level contribution of about 5 ug/dL from water is predicted for this infant.

  The real questions become: To what extent would corrosion control treatment be expected to reduce contributions to blood lead levels from water, and what health benefits, if any, may result?

- Optimal corrosion control treatment would provide only minimal reduction in the contributions from water to an individual's blood lead level.

  Consider again the highly unlikely but possible exposure scenario of an individual infant consistently consuming standing water with 49 ug/L of lead (the 90th percentile standing lead level in Portland's Tier 1 homes in 1992). Optimal corrosion control treatment would be expected to reduce a "before treatment" water lead level of 49 ug/L by about 70% to 15 ug/L; this would correspond to a minimal decrease in blood lead contribution for an infant from about 5 to 4 ug/dL. In such cases, other interventions such as tap flushing or replacement of lead bearing materials in premise plumbing would be needed.

- Even with optimal corrosion control treatment, the predicted percentage of the population with elevated blood lead levels would essentially remain unchanged.

  The population-based model predicts that the amount by which the "before treatment" and predicted "after treatment" blood lead distributions in Portland differ would be < 1 ug/dL. For example, consider the conservative exposure scenario in which Portland infants consistently consume a mix of running (75%) and standing (25%) water. The "before treatment" median, 90th, and 95th percentile blood lead levels for this group are estimated at 3.8, 8.6, and 11.0 ug/dL, respectively. With corrosion control treatment involving pH adjustment to 7.5-9.5, each of these percentiles is predicted to decrease by only 0.3-0.5 ug/dL.

- There is very little difference between the blood lead level reductions that can be expected with limited corrosion control treatment (pH adjustment to 7.5) as compared to LCR-defined optimal corrosion control treatment (pH adjustment to 9.5).

  Population-based modeling predicts that the amount by which the predicted blood lead distributions for "limited treatment" and "optimal treatment" scenarios differ is typically by <= 0.2 ug/dL.

  Individual-based modeling predicts that the differences in reductions of individual blood lead level contributions from water with a limited level of corrosion control treatment (pH adjustment to 7.5) as compared to LCR-defined optimal treatment (pH adjustment to 9.5) are typically less than 1 ug/dL.

- While corrosion control treatment of Portland's Bull Run water supply would be expected to reduce lead in water by 40-70% as compared to "no treatment" level, the type and extent of any resulting health benefits are much less certain.

  For children with blood lead levels already below the 10 ug/dL level of concern, there is currently no conclusive evidence that reducing blood lead levels further, especially by the small levels associated with corrosion control treatment, would provide any health benefits. Again, Portland is considered a community in which there is a low-prevalence of elevated blood levels; and even with optimal corrosion control treatment; the predicted percentage of the population with elevated blood lead levels would essentially remain unchanged.

  For children with blood lead levels that are well above the 10 ug/dL level of concern, corrosion control is not likely to reduce these levels to below 10 ug/dL.

- Interventions to reduce lead exposures should be targeted at those exposure pathways most significantly contributing to a child's total exposure.

  Since lead-based paint is the most commonly identified source of elevated blood lead levels in Portland, interventions should be focused on this source.

## Planned Next Steps

The results of this study predict a very limited level of effectiveness of corrosion control treatment in reducing the community's blood lead levels. Even with the unrealistic worst case scenario that the entire community consumes only standing water, reduction in blood lead levels in the community and in the reduction the percentage of people with elevated blood lead levels are minimal. Additionally, the difference between the effects predicted with optimal corrosion control as compared to limited corrosion control are even smaller.

These findings support the Portland Water Bureau's proposal to implement a Lead Hazard Reduction Plan (LHRP) that would:

- Prevent lead poisoning before it occurs (primary prevention); and
- Focus efforts on those persons who are at most risk to significant lead exposure and on those lead source and exposure pathways that would be expected to have the greatest impact on reducing a child's body lead burden.

As part of the LHRP, limited corrosion control treatment provides pH adjustment up to 7.5 to meet the copper action level and reduce lead levels in water by an estimated 40%. The savings in capital and operating costs associated with operating at a pH of 7.5 versus 9.5 would be used to fund other targeted interventions that are designed to achieve better public health protection from lead exposure.

# Section 1 - Introduction

The City of Portland is the largest supplier of drinking water in Oregon. Portland's Bureau of Water Works (Water Bureau) provides water to about 500,000 people within its service area, and to an additional 300,000 people in the metropolitan area through wholesale water sales to 19 public water systems. The principal source of water is the Bull Run watershed, a protected, unfiltered surface water supply. Portland also has a well field on the south shore of the Columbia River, which is used if necessary to meet summer peak and emergency demands.

## 1.1 Purpose of this Investigation

The Lead and Copper Rule (LCR) requires water systems to monitor lead and copper levels in samples from customers' taps that are likely to be at highest risk for elevated levels of these metals. Two rounds of initial monitoring were required in 1992. The 90th percentile lead level was 44 ug/L for the first round, and 53 ug/L for the second round. The Lead and Copper Rule lead action level of 15 ug/L at the 90th percentile was exceeded, triggering implementation of a mandated public education program.

The Water Bureau became interested in a number questions raised by these results, including:

- What is the distribution of lead levels in drinking water throughout Portland, not just in "highest-risk" homes?
- How do these water lead levels contribute to blood lead levels?
- How much would water lead levels and blood lead levels be reduced with corrosion control treatment?
- What are the associated health effects issues?

In June 1994, the Water Bureau completed its corrosion control study as required by the LCR. This study (MW and EES, 1994) indicates that minimizing lead and copper in Bull Run water would involve increasing pH in the distribution system from levels of about 6.8 after chlorination and ammoniation to pH 9.0-9.5, and also increasing alkalinity from levels of 6-12 mg/L to at least 25 mg/L as $CaCO_3$.

Also in June 1994, the Portland City Council, in accordance with recommendations from the citizens' Water Quality Advisory Committee, and the Water Managers Advisory Board (managers of water systems purchasing Bull Run water), directed the Water Bureau to investigate alternatives for LCR compliance. The resulting report (EES, 1995) included development of preliminary models to estimate the effects of various interventions on lead exposure through drinking water, as indicated by predicted changes in blood lead levels.

The most significant conclusions of this study were:

- Drinking water is not a major route of lead exposure in the Portland area.
- Although water treatment would provide some reduction of lead and copper exposure through drinking water in the community, water treatment alone would not sufficiently reduce the potential for significant lead exposure in some homes with very elevated levels of lead in standing water.
- The most significant source of lead exposure in the Portland metropolitan area is lead-based paint, and efforts focused on preventing exposures from this source could provide a significant health benefit to the community.

The purpose of this study and report is to estimate the effects of corrosion control treatment on lead exposure through drinking water and to describe what, if any, health benefits may be realized as a result of water treatment.

## 1.2 Model Approaches to Estimate Lead Exposure Through Drinking Water

Two modeling approaches were used: individual-based models and population-based models. Individual-based modeling was used to estimate the contribution from lead in water to the blood lead level of an individual infant, child, or adult living in Portland before corrosion control treatment and after implementation of various levels of corrosion control treatment.

Population-based modeling was used to compare the distribution of blood lead levels before corrosion control treatment for infant, child and adult populations within Portland to predicted distributions after implementation of various levels of corrosion control treatment.

The models require various types of input, including:

- the distribution of water lead levels before corrosion control treatment, described in Section 2;
- the anticipated reduction of water lead levels with corrosion control treatment, also described in Section 2;
- the distribution of blood lead levels before corrosion control treatment, described in Section 3; and
- coefficients for the contribution of water lead to blood lead levels, described in Section 4.

These models, described in detail in Sections 4 and 5, make use of best available input data. Results are not intended to be interpreted on a precise quantitative basis, but rather should be used as relative indicators of potential reductions in blood lead levels under various lead exposure scenarios.

# Section 2 - Distribution of Drinking Water Lead Levels in Portland

## 2.1 Bull Run Water Quality

Water from the Bull Run watershed is characterized by naturally low levels of mineralization typical of surface waters fed from rapid rainfall runoff in mountainous areas of the Pacific Northwest. Untreated Bull Run water has relatively low levels of pH (median value 7.0), alkalinity (median value 7.0 mg/L as $CaCO_3$), and is poorly buffered to resist changes in pH. These characteristics make it relatively aggressive toward metallic materials present in utility distribution and premise piping systems.

## 2.2 Sources of Lead in Drinking Water

There is no detectable amount of lead (< 0.001 mg/L) in Portland's source water. Lead enters drinking water primarily as a result of corrosion of home and building plumbing materials. The most common sources include lead-soldered joints in copper pipe, and faucets and other fixtures made from lead-bearing brass. The lead content of solder in water systems has been limited to less than 0.1% in Oregon since 1985 (prior to that time, solder typically contained 50% lead). Brass used in plumbing fixtures may contain up to 8% lead and still be called "lead-free". Copper pipe has been the most commonly used type of household water pipe material since the 1960s.

There are no lead water mains or service lines within the City of Portland’s service area or within the service areas of its wholesale water customers. There are, however, some short sections of lead pipe, called pigtails, connecting mains and customer service lines, still existing in the distribution system. The Portland Water Bureau has been removing these lead service line connectors from its distribution system since 1984; the approximately 1500 remaining lead service line connectors are scheduled to be removed by July 1998.

When corrosive water stands in contact with sources of lead for several hours, these metals can dissolve in drinking water. This means that the first water drawn from the tap after at least a 6-8 hour standing period (called a standing sample) can contain elevated levels of these metals. Running samples (samples taken from the tap after water has run for a short time) have significantly lower levels of metals than standing samples. In addition to standing time, there are many other factors that influence the pick up of metals by the water including pH, alkalinity, temperature and presence of orthophosphate.

## 2.3 Lead in Drinking Water Data

### 2.3.1 Lead and Copper Rule - Initial Monitoring Period Data

The Lead and Copper Rule (LCR) requires water systems to collect 1-liter standing water samples from Tier 1, 2 or 3 sites, which are samples from customers' taps that are likely to be at highest risk for elevated levels of lead and/or copper in drinking water. Results of initial monitoring required in 1992-93 for Portland and other Bull Run water systems are shown in Table 2-1.

### 2.3.2 Customer Requests for Free Lead-in-Water Analysis

The Portland Water Bureau has a policy of providing water testing for lead free of charge to its customers upon request. Standing samples, which are most likely to contain elevated lead and copper levels, are most frequently collected; however, sometimes both a standing and a running sample tap sample are collected. This set of data, summarized Table 2-2, includes samples from homes of various ages located throughout Portland. Although it does not represent a true random sample, it is the best data set available to describe the occurrence of lead in drinking water in the City of Portland.

**Table 2-1**
**Lead and Copper Rule Tap Monitoring - City of Portland and Contract Holders**
**Results of Initial Monitoring in 1992-93**

| Water System Name | Population (1) | LCR System Size | Req'd no. samples | 1st round - Initial monitoring period | Actual no. of samples | 90th %tile lead value (mg/L) | 2nd round Initial monitoring period (2) | Actual no. of samples | 90th %tile lead value (mg/L) | 90th %tile lead value, pooled data from both initial monitoring periods |
|---|---|---|---|---|---|---|---|---|---|---|
| Burlington WD | 390 | Small | 10 | Jul-Dec 93 | 10 | 0.011 | | | | |
| GNR Corp | 72 | Small | 5 | Jul-Dec 93 | 5 | 0.008 | | | | |
| Gresham, City of | 35,000 | Medium | 60 | Jul-Dec 92 | 60 | 0.041 | | | | |
| Lake Grove WD | 3,300 | Medium | 20 | Jul-Dec 93 | 20 | 0.062 | | | | |
| Lorna WD | 200 | Small | 10 | Jul-Dec 93 | 10 | 0.015 | | | | |
| Lusted | 1,300 | Small | 20 | Jul-Dec 93 | 20 | 0.007 | | | | |
| Palatine Hill WD | 1,500 | Small | 20 | Jul-Dec 93 | 21 | 0.075 | | | | |
| Pleasant Home WD | 1,200 | Small | 20 | Jul-Dec 93 | 20 | 0.030 | | | | |
| Portland, City of | 460,000 | Large | 100 | Jan-Jun92 | 126 | 0.044 | Jul-Dec 92 | 125 | 0.053 | 0.049 |
| Powell Valley Road WD | 32,000 | Medium | 60 | Jul-Dec 92 | 60 | 0.035 | | | | |
| Raleigh WD | 4,000 | Medium | 20 | Jul-Dec 92 | 30 | 0.034 | | | | |
| Rockwood WD | 43,000 | Medium | 60 | Jul-Dec 92 | 61 | 0.037 | | | | |
| Skyview Acres WD | 47 | Small | 5 | Jul-Dec 93 | 5 | 0.022 | | | | |
| Tigard, City of | 37,350 | Medium | 60 | Jul-Dec 92 | NA (3) | NA (3) | NA (3) | NA (3) | NA (3) | |
| Tualatin, City of | 17,450 | Medium | 60 | Jul-Dec 92 | 60 | 0.043 | | | | |
| Tualatin Valley WD | 144,980 | Large | 100 | Jan-Jun92 | 102 | 0.028 | Jul-Dec 92 | 102 | 0.029 (4) | 0.029 |
| Valley View WD | 950 | Small | 10 | Jul-Dec 93 | 9 | 0.039 | | | | |
| West Slope WD | 12,000 | Medium | 60 | Jul-Dec 92 | 75 | 0.039 | | 79 | 0.037 | |
| **POOLED DATA FOR ALL SYSTEMS:** | | | | **Total number of samples: 900** | | | **90th percentile lead value: 43 ug/L** | | | |

(1) Source: OHD

(2) OHD waived requirement for 2nd round of monitoring for small/medium systems that exceeded lead and/or copper action levels in first round of monitoring

(3) Tigard not using Bull Run water during initial monitoring period

(4) Sources of water other than Bull Run in use at the time of monitoring

**Table 2-2**
**Lead Levels at Customers' Taps in Portland [1]**
**Customer Request Data Base**

| | RUNNING Samples[2] | STANDING Samples[3] |
|---|---|---|
| 50th percentile | < 1 ug/L | 4 ug/L |
| 90th percentile | 3 ug/L | 14 ug/L |
| 95th percentile | 5 ug/L | 23 ug/L |
| 98th percentile | 9 ug/L | 44 ug/L |
| Percentage of samples that meet the FDA's limit of 5 ug/L of lead in bottled water | 95% | 69% |
| Number of samples | 2309 | 5434 |

ug/L: micrograms per liter (parts per billion)

(1) Portland customer requests for free lead in water analysis, 1986 - 97.
(2) Samples taken from residential kitchen or bathroom taps after water has been allowed to flow for at least one minute.
(3) Samples taken from residential kitchen or bathroom taps that have stood in contact with home plumbing materials for 6-12 hours.

Table 2-3 shows the occurrence of lead in drinking water by age of home as determined by correlating Portland's several recent years of water quality data with building records. Statistical tests indicate that the homes where the highest lead values were observed were in those homes built in 1980-1984 and in 1930-39. Homes built in 1980-84 are likely to have to have "new" lead-based solder in premise water plumbing. Homes built before 1930 and all other home age categories were not significantly different from the total sample set. Homes built before 1930 comprise nearly one-third of the homes in Portland.

**Table 2-3**
**Lead Levels at Customers' Taps in Portland by Year Home Built**
**Standing Samples [1]**

| Year Home Built | Number of samples | Median (50th percentile) (ug/L) | 90th percentile (ug/L) | % of Homes in Multnomah County [4] |
|---|---|---|---|---|
| Before 1930 [3] | 466 (43%) | 6 | 24 | 31% |
| 1930-1939 [3] | 44 (4%) | 5 | 46 | 3% |
| 1940-1949 [3] | 70 (7%) | 6 | 28 | 10% |
| 1950-1959 [3] | 71 (7%) | 4 | 19 | 14% |
| 1960-1969 [3] | 54 (5%) | 8 | 34 | 13% |
| 1970-1979 [3] | 72 (7%) | 10 | 32 | 17% |
| 1980-1984 [2][3] | 264 (25%) | 7 | 49 | 5% |
| 1985-1995 [3] | 17 (2%) | 4 | 14 | 7% |
| **Total** | **786 (100%)** | | | **100%** |

(1) Samples taken from residential kitchen or bathroom taps that have stood in contact with home plumbing materials for at least 6 hours.
(2) Samples from "Tier 1" homes in collected in 1992 as required by the LCR.
(3) Customer requests for free lead in water analysis, 1992-1994.
(4) Multnomah County Tax Assessor data

## 2.4 Distributions of Drinking Water Lead Levels Used in Models

The distributions of lead in running and standing samples from Portland's Customer Request Database described in Table 2-2 were used in the individual- and population-based exposure models.

The running water distribution is comprised of samples taken from residential taps after water had been allowed to flow for at least one minute. This distribution represents the lead content of water drawn from taps frequently throughout the day that has had no significant standing contact time with home plumbing materials, and represents a "best-case" (least lead) consumption scenario.

The standing water distribution is comprised of first-draw 1-liter samples taken from residential taps that have stood in contact with home plumbing materials for 6-12 hours. This distribution represents the lead content of water drawn from taps first drawn in the morning and/or after returning from work. Although some people may occasionally consume standing water, it is highly unlikely that an individual's water consumption consist's solely of standing water.

A hybrid distribution of water lead levels was calculated to represent a consistent consumption pattern of 75% running water and 25% standing water. This hybrid distribution is a weighted combination of the standing and running distributions. This is intended as a conservative consumption scenario that recognizes that both running and standing water are probably consumed.

## 2.5 Estimated Reduction of Lead in Drinking Water with Corrosion

## Control Treatment

Three corrosion control treatment alternatives were selected for modeling that involve pH adjustment to the following ranges:

| pH in distribution system | Treatment Condition |
|---|---|
| <= 7.0 | Baseline: no corrosion control treatment |
| 7.0-7.5 | Limited corrosion control treatment |
| 8.0-8.5 | Moderate corrosion control treatment |
| 9.0-9.5 | LCR-defined "optimal" treatment to minimize lead and copper |

A number of sources of information were evaluated to estimate the extent to which pH adjustments in these ranges would result in reduced lead levels in drinking water. These include theoretical solubility calculations, bench scale electrochemical and pipe loop testing of Bull Run water, and analogous system data, as shown in Table 2-4. The estimated extent of water lead reduction for various levels of corrosion control treatment are also shown in Table 2-4. Estimated reductions are expressed in terms of percent reductions in water lead levels from levels associated with no corrosion control treatment.

**Table 2-4**
**Evaluation of Potential Reductions in Water Lead Levels**
**for Various Levels of pH Adjustment (EES, 1995) (MW and EES, 1996)**

| **Lead Reduction** | **pH 7-7.5** | | **pH 8-8.5** | | **pH 9-9.5** | |
|---|---|---|---|---|---|---|
| Source of Information | Median | 90 %tile | Median | 90 %tile | Median | 90 %tile |
| Theoretical Solubility (1) | 38-68% | | 70 % | | 89 % | |
| Bench Tests (2) | 25-86% | | 55-87% | | 25-79% | |
| Phase 1 Pilot Studies (3) | | | | | | |
| Lead Solder | | | 50% | 67% | 68% | 56% |
| Brass | | | 64% | 86% | 92% | 95% |
| Phase 2 Pilot Studies (3) | | | | | | |
| Lead Solder | | | | | 50-66% | 31-69% |
| Brass | | | | | 92-95% | 6-65% |
| Phase 3 Pilot Studies (3) | 50-75% | | 50-74% | | 50-95% | |
| Analogous Systems | | | | | | |
| Seattle | | | 61-68% | 64% | | |
| GVWD Demonstration | -16 % | | 16% | | | |
| Bellingham | | | 61% | 35% | | |
| WITAF Study | | 74% | | 80% | | 76% |
| **Used for Evaluation** | **40%** | **40%** | **60%** | **60%** | **70%** | **70%** |

Notes:
(1) Based on solubility calculations.
(2) Electrochemical test data by University of Washington for Portland Water Bureau.
(3) Pipe rack testing data by Portland Water Bureau

# Section 3 - Distribution of Blood Lead Levels in Portland

## 3.1 Lead in Blood

This study sought to identify the existing distribution of blood lead levels in the Portland area. This distribution was used in the population-based exposure model as a baseline from which predicted changes in blood lead level due to corrosion control treatment could be estimated.

## 3.2 National Health and Nutritional Examination Surveys (NHANES) Data

At the time the Lead and Copper Rule was developed, the best available study of blood lead levels in the United States was the National Health and Nutrition Examination Survey II (NHANES II) (Brody and others, 1994). The NHANES II study included measurement of blood lead levels in over 40,000 random samples collected from 1978 to 1983 from people across the country. Results indicated that the median blood lead level was 12.8 ug/dL and that nearly 80% of Americans had blood lead levels above 10 ug/dL, the current level of concern, as shown in Figure 3-1. The preamble to the Lead and Copper Rule states that “because many children now have blood lead levels above the level of concern, EPA’s policy goal continues to be that drinking water should contribute minimal additional lead to existing body burdens of lead” (USEPA, 1991).

In 1994, the results of the first phase of the follow-up study, NHANES III, were published (Brody and others, 1994). The NHANES III study included blood lead level measurements collected from 1988 to 1991. (The Lead and Copper Rule was promulgated in 1991). Results indicated that the median blood lead level had dropped from 12.8 ug/dL to 2.8 ug/dL and that less than 10% of Americans had blood lead levels above the level of concern, a tremendous reduction in blood lead levels from 1978-1983 levels, as shown in Figure 3-2. This dramatic reduction in blood lead levels is primarily attributed to the increased use of non-leaded gasoline (Pirkle and others, 1994). NHANES III summary statistics are shown in Table 3-1.

**Figure 3-1**

**US Blood Lead Levels**
**NHANES II Study**
**Measured in 1978-1983 (from Brody, and others, 1994)**

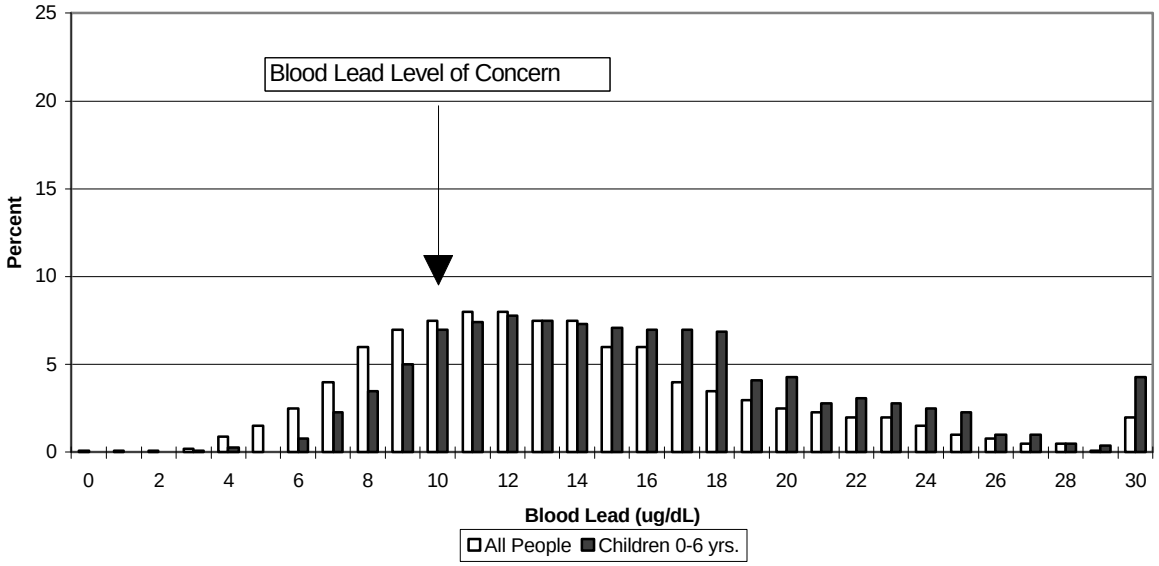



**Figure 3-2**

**US Blood Lead Levels**
**NHANES III Study**
**Measured in 1988-1991 (from Brody, and others, 1994)**

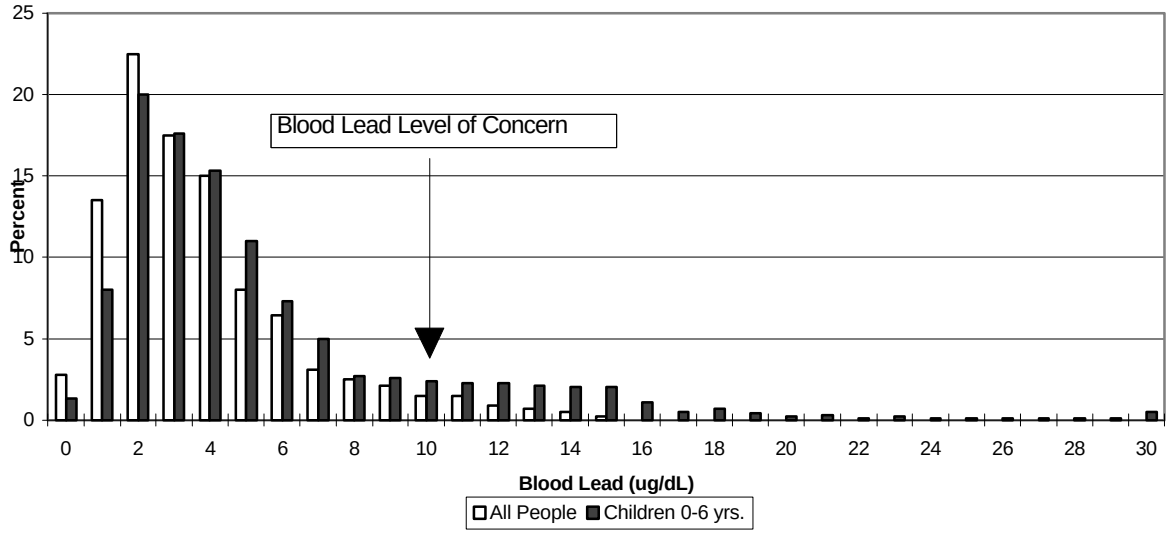


**Table 3-1**
**NHANES III Summary Statistics**
**(Brody and others, 1994)**

| Years (1988-1991) | No. | Geometric Mean (ug/dL)* | 95% Confidence Interval (ug/dL) | Percentiles (ug/dL) 5th | 10th | 25th | 50th | 75th | 90th | 95th |
|---|---|---|---|---|---|---|---|---|---|---|
| All Persons | 12119 | 2.8 | 2.7-3.0 | <1.0 | 1.0 | 1.8 | 3.0 | 4.8 | 7.3 | 9.4 |
| Ages 1-5 | 2234 | 3.6 | 3.3-4.0 | 1.1 | 1.5 | 2.2 | 3.7 | 5.9 | 9.6 | 12.2 |
| Ages 6-19 | 2963 | 1.9 | 1.7-2.2 | <1.0 | <1.0 | 1.3 | 2.1 | 3.5 | 5.4 | 7.4 |
| Ages 20-74 | 6922 | 3.0 | 2.8-3.2 | <1.0 | 1.2 | 2.0 | 3.2 | 5.0 | 7.4 | 9.5 |
| Males | 6051 | 3.7 | 3.5-3.9 | 1.2 | 1.6 | 2.4 | 3.8 | 5.8 | 8.7 | 10.9 |
| Females | 6068 | 2.1 | 2.0-2.2 | <1.0 | <1.0 | 1.4 | 2.3 | 3.8 | 5.7 | 7.4 |
| Non-Hispanic Whites | 4337 | 2.7 | 2.2-2.8 | <1.0 | 1.0 | 1.7 | 2.9 | 4.5 | 6.8 | 8.9 |
| Non-Hispanic Blacks | 3274 | 3.5 | 3.3-3.9 | <1.0 | 1.3 | 2.2 | 3.7 | 5.9 | 9.3 | 12.1 |
| Non-central City | 7495 | 2.7 | 2.5-2.8 | <1.0 | 1.0 | 1.8 | 3.0 | 4.6 | 6.9 | 8.9 |
| Central City, <1 million | 2909 | 2.9 | 2.5-3.4 | <1.0 | 1.0 | 1.8 | 3.0 | 5.2 | 8.3 | 10.4 |
| ≥1 million | 1379 | 3.9 | 3.6-4.3 | 1.3 | 1.8 | 2.5 | 4.0 | 6.1 | 9.9 | 13.2 |
| Income level, Low† | 4106 | 3.4 | 3.1-3.8 | <1.0 | 1.3 | 2.1 | 3.6 | 5.8 | 9.4 | 11.8 |
| Income level, mid† | 4050 | 2.7 | 2.6-2.9 | <1.0 | 1.0 | 1.7 | 2.9 | 4.7 | 7.1 | 9.1 |
| Income level, high† | 2781 | 2.5 | 2.4-2.7 | <1.0 | <1.0 | 1.7 | 2.8 | 4.3 | 6.3 | 8.0 |

* For each grouping, the geometric means from NHANES II and NHANES III phase are statistically different (P>.01).
† Income level was defined by poverty income ratio (PIR) categorized as low (0<PIR<1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00)

## 3.3 Oregon Childhood Lead Poisoning Prevention Project (OCLPPP) Data

The OCLPPP is an on-going project coordinated by the Oregon Health Division (OHD) and funded by a grant from the Centers for Disease Control (CDC). A major component of the project is blood lead level screening of children in four Oregon counties, including Multnomah County. The City of Portland is located in Multnomah County.

Table 3-2 is a summary of OCLPPP blood lead screening data for children 0-6 years of age in Multnomah County collected from 1992 through 1994. Children were screened in elementary schools and County Health Clinics and other community locations. Because of the inclusion of County Health Clinic samples, which includes a large percentage of low income families, this distribution of blood lead data may be somewhat higher than would be observed in a completely randomized sample of the population.

**Table 3-2**
**1992-94 OCLPPP Blood Lead Levels by Year Home Built**
**Children in Multnomah County 0-6 Years of Age**

| | Blood Lead Level (ug/dL) | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| **Year Home Built** | **3 or Lower** | **4** | **5** | **6** | **7** | **8** | **9** | **10-14** | **15 or higher** | **Total** |
| Pre-1930 | 360 | 125 | 98 | 69 | 71 | 34 | 31 | 56 | 39 | 883 |
| 1930-1939 | 45 | 3 | 8 | 7 | 6 | 3 | 1 | 4 | 0 | 77 |
| 1940-1949 | 104 | 35 | 19 | 12 | 2 | 7 | 3 | 4 | 2 | 188 |
| 1950-1959 | 101 | 37 | 21 | 14 | 14 | 5 | 6 | 5 | 1 | 204 |
| 1960-1969 | 110 | 46 | 31 | 13 | 8 | 4 | 15 | 14 | 7 | 268 |
| 1970-1979 | 187 | 52 | 45 | 28 | 22 | 19 | 7 | 16 | 2 | 378 |
| 1980-1984 | 19 | 4 | 3 | 3 | 3 | 3 | 0 | 1 | 0 | 36 |
| 1985-1989 | 11 | 3 | 0 | 1 | 1 | 1 | 0 | 0 | 0 | 17 |
| 1990-1994 | 14 | 6 | 3 | 1 | 1 | 0 | 0 | 3 | 2 | 30 |
| **Total** | **971**<br>**46.66%** | **311**<br>**14.94%** | **228**<br>**10.96%** | **148**<br>**7.11%** | **128**<br>**6.15%** | **76**<br>**3.65%** | **63**<br>**3.03%** | **103**<br>**4.95%** | **53**<br>**2.55%** | **2081**<br>**100%** |

The Oregon Health Division (OHD) conducted an analysis of the relationship between elevated blood lead levels and year of construction of homes in which these levels occur. The data set analyzed included OCLPPP data and other reported elevated blood lead data from Multnomah County. The data in Table 3-3 and Figure 3-3 show strong positive relationship between occurrence of elevated blood lead level and the probability of living in a home built before 1930. About 1 out of 6 of those tested who were living in homes built before 1930 had elevated blood lead levels ($\geq$10 ug/dL), as compared to about 1 out of 15 tested living in homes built in 1930 or thereafter. OHD's analysis also suggests that various subpopulations may be at higher than average risk: for example, children 2-3 years old, African-American children, and Hispanic children.

**Table 3-3**
**Occurrence of Elevated Blood Lead Levels for Homes Built Before and After 1930**
**Multnomah County**

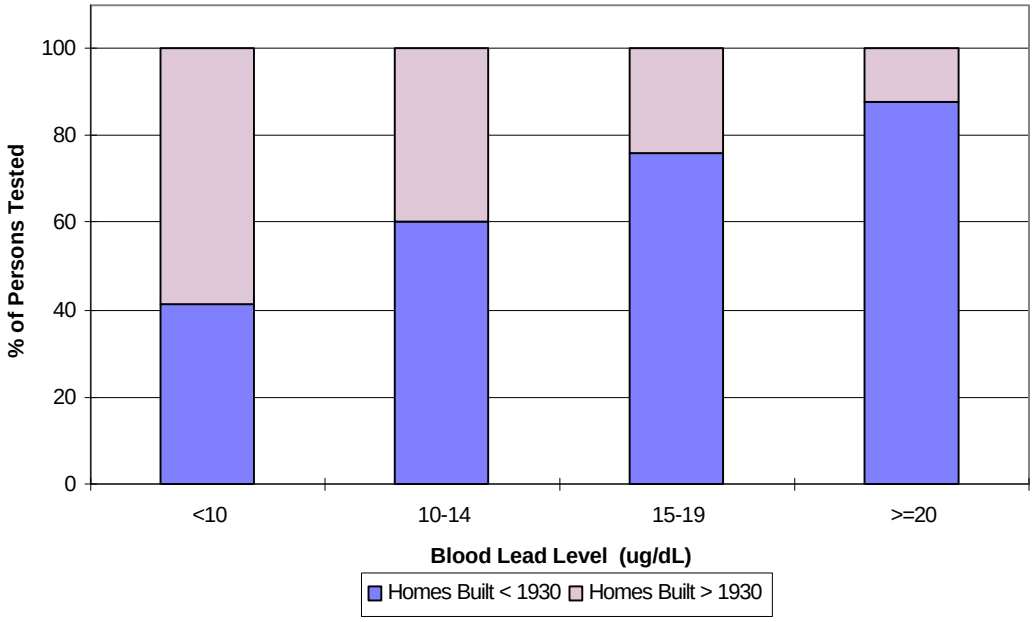
| Blood Lead Level (ug/dL) | Percentage of persons tested living in homes built before 1930 with blood lead level listed in 1st column | Percentage of persons tested living in homes built in 1930 or after with blood lead level listed in 1st column |
|---|---|---|
| < 10 | 41 | 59 |
| 10-14 | 60 | 40 |
| 15-19 | 76 | 24 |
| 20 or more | 88 | 12 |
| | | |
| Chances of having an elevated blood lead level, EBLL: | | |
| ≥ 10 ug/dL | 1 in 6 (17.6%) | 1 in 15 (6.8%) |
| ≥ 15 ug/dL | 1 in 13 (7.6%) | 1 in 68 (1.5%) |
| ≥ 20 ug/dL | 1 in 34 (2.9%) | 1 in 305 (0.3%) |

n= 2179

**Figure 3-3**
**Occurrence of Elevated Blood Lead Levels**
**For Homes Built Before and After 1930**
**Multnomah County**

## 3.4 Sources of Lead Exposure in Cases of Elevated Blood Lead Levels

Medical laboratories in Oregon are required to report cases of elevated blood lead



levels (EBLLs) of 10 ug/dL or higher to the Oregon Health Division (OHD). Since 1993, the Multnomah County Health Department (MCHD) has conducted about 120 follow-up investigations of EBLLs in Multnomah County and the Portland metropolitan area. These investigations indicate that lead-based paint is the most likely source of exposure for 70% of the EBLLs of at least 15 ug/dL, and for 80% of the EBLLs of at least 20 ug/dL (OHD, 1997). For the remaining cases of EBLLs where the most likely sources of lead exposure could be identified, the sources included occupational or hobby related sources, sources from the country of origin of recent immigrants, and water (1 case). As part of 24 of these follow-up investigations of EBLLs occurring within Portland, the Water Bureau was requested to analyze tap water samples for lead. For this group of 24 samples, the median lead level in standing samples is 2 ug/L and the 90th percentile lead level is 13 ug/L (PWB, 1997). These concentrations are consistent with the median lead level in standing samples in Portland which is estimated at 4 ug/L and the 90th percentile concentration which is estimated at 14 ug/L. These data suggest that water was an insignificant or minor exposure pathway in these cases.

The observation that lead-based paint is the most commonly identified source of EBLLs in Multnomah County is consistent with the CDC's statement that lead-based paint is the most common high-dose source of lead exposure for children (CDC, 1991). Also, the EPA has estimated that, for a typical 2-year-old child living in an urban environment or in a non-urban house with interior lead-based paint, household dust and soil account for more than 90 percent of the child's daily intake of lead (USEPA, 1995).

## 3.5 Distribution of Blood Lead Levels Used in Models

As part of the study to evaluate alternatives for LCR compliance (EES, 1995), blood lead level distribution data were evaluated in consultation with the Occupational, Environmental, and Injury Epidemiology (OEI-EPI) section of the Oregon Health Division (OHD). It was concluded that the best available data to characterize the existing distribution of blood lead levels in Portland is:

- For infants and children less than 6 years of age: Oregon Childhood Lead Poisoning Prevention Project (OCLPPP) screening data from Multnomah County, 1992 through 1994 (Table 3-2).
- For all others: National Health and Nutrition Examination Survey (NHANES) III, Phase I National Summary Statistics, 1988 through 1991 (Table 3-1). These national data were used due to a lack of local data for population age groups other than children. Blood lead testing of adults in Oregon and elsewhere is generally limited to cases of suspected lead poisoning, and results are only reported when blood lead levels are above the 10 ug/dL level of concern.

Table 3-4 is a summary of the blood lead level distributions for these two sets of data.

**Table 3-4**
**Summary of Best Available Data to Characterize Blood Lead Levels in Portland**

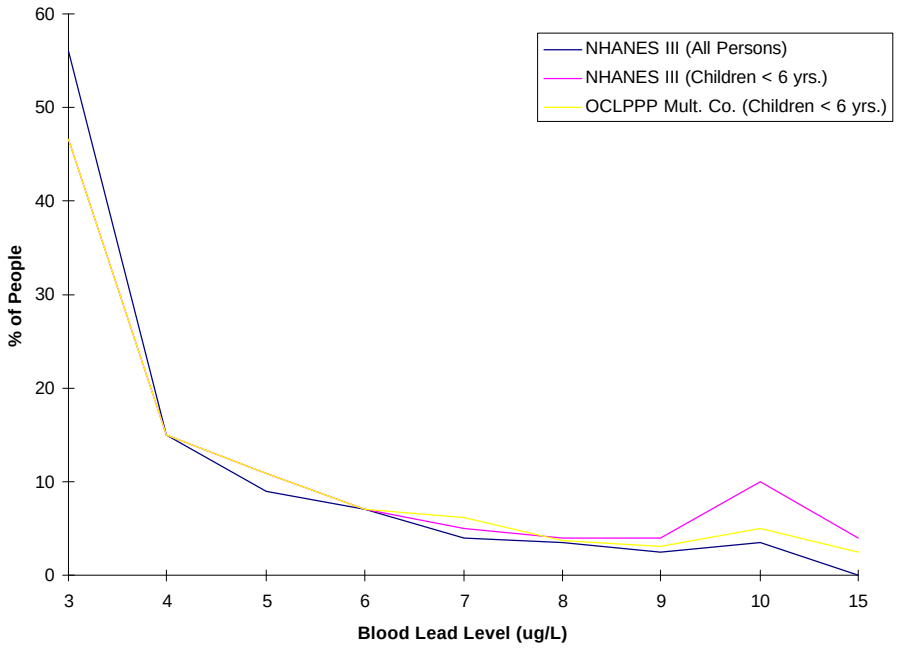
| Statistic | Infants: OCLPPP (1) | Children: OCLPPP (1) | Adults: NHANES III (2) |
|---|---|---|---|
| 5th percentile | <2.0 ug/dL | <2.0 ug/dL | 0.5 ug/dL |
| 50th percentile (median) | 3.8 ug/dL | 3.6 ug/dL | 3.0 ug/dL |
| 70th percentile | 5.1 ug/dL | 5.0 ug/dL | 4.4 ug/dL |
| 90th percentile | 8.6 ug/dL | 8.4 ug/dL | 7.4 ug/dL |
| 95th percentile | 11.0 ug/dL | 10.8 ug/dL | 9.5 ug/dL |
| Number of samples | 2081 | 2081 | 40,000 |
| % of people with elevated blood lead levels ($\geq$ 10 ug/dL) | 5.9% | 5.9% | 4.6% |

(1) Oregon Childhood Lead Poisoning Prevention Project, Multnomah County, 1992 through 1994, children 0-6 years of age. Children tested were County clinic patients or were at targeted community screening locations.

(2) National Health and Nutrition Examination Survey, Phase III, 1988-91 (Brody and others, 1994).

As shown on Figure 3-4, NHANES III data correlate well with OCLPPP data. A paired t-test was conducted to compare OCLPPP and NHANES III data for children less than 6 years of age. The test indicates that there is no significant difference between these two distributions of blood lead data.

# Figure 3-4
# Comparison of NHANES III and Multnomah County OCLPPP Blood Lead Level Distributions



# Section 4 - Individual-based Modeling of Lead Exposure through Drinking Water

## 4.1 Description of the Model

Individual-based modeling was used to estimate the contribution from water with a given lead level to the blood lead level of an individual infant, child, or adult. For a given water lead level of interest, the contribution to an individual's blood lead level was calculated by multiplying the water lead level by coefficients relating lead levels of water consumed to blood lead contributions.

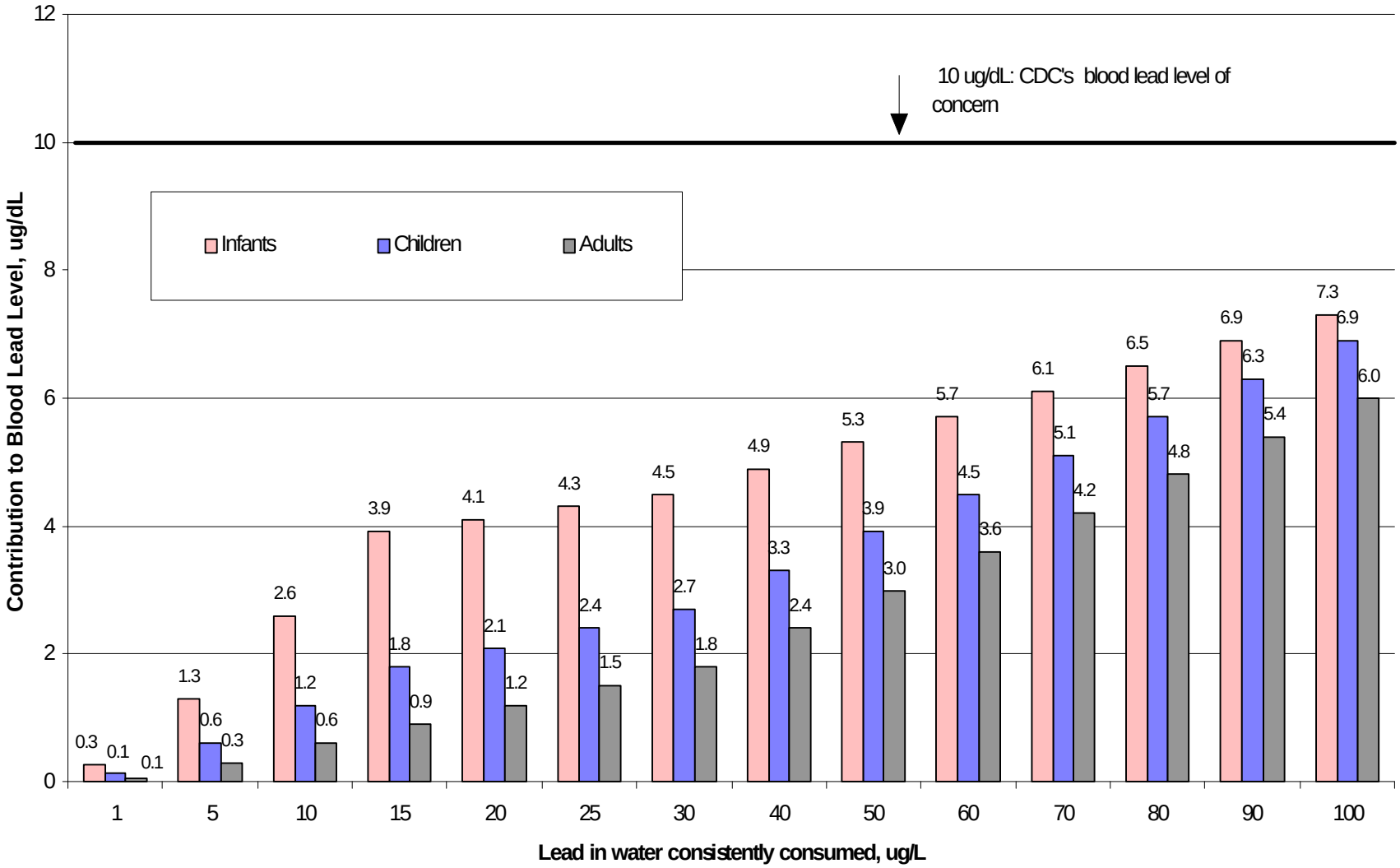
In the preamble to the Lead and Copper Rule, the EPA cites the best available studies for estimating the relationship between blood lead and water lead levels (USEPA, 1991). The EPA's analysis of several health effects studies found a nonlinear relationship between drinking water lead and blood lead in children. The relationship was best described by a classic piecewise dose response function with different coefficients at different water lead concentrations. At higher drinking water lead concentrations, less lead is generally adsorbed in the blood than at lower drinking water lead concentrations.

**Table 4-1**
**Summary of Best Available Coefficients for Estimating the Relationship Between Water Lead and Contributions to Blood Lead Levels (USEPA, 1991)**

| For: | Drinking Water Lead <=15 ug/L | Drinking Water Lead >15 ug/L |
|---|---|---|
| Children < 6 months | 0.26 ug/dL blood per ug/L water | 0.04 ug/dL blood per ug/L water |
| Children ≥ 6 months to < 6 years | 0.12 ug/dL blood per ug/L water | 0.06 ug/dL blood per ug/L water |
| Adults | 0.06 ug/dL blood per ug/L water | 0.06 ug/dL blood per ug/L water |

As an example of how these coefficients were applied, consider the exposure scenario of an infant consistently consuming water with a lead level of 25 ug/L. The contribution to the infant's blood level (child less than 6 months) is predicted to be (15 * 0.26) + ( (25-15) * 0.04) ) = 4.3 ug/dL. A set of predicted contributions from water to an infant's, child's, and adult's blood lead level are shown in Figure 4-1. These predicted contributions are "generic" in the sense that they are presumably relevant for any individual who consistently consumes water with a given lead level, regardless of its source.

**Figure 4-1**
**Estimated Contribution of Water Lead to Blood Lead Levels**
**Individual-Based Modeling**



Contributions from water to an individual's blood lead level were calculated for specific water lead levels of interest within the range of 1-50 ug/L. These are shown in Tables 4-2 through 4.5. This range was chosen because:

1) the median lead level in running water in Portland homes is estimated at < 1 ug/L (Table 2-2); and

2) the 90th percentile lead level in standing water in Portland's "Tier 1" homes (those at highest-risk for elevated water lead levels) during the initial 1992 LCR-required monitoring period was 49 ug/L (see Table 2-1).

Predicted contributions from water to an individual's blood lead level for lower water lead levels anticipated with corrosion control treatment were also calculated. These are shown in Tables 4-2 through 4-5. Three levels of corrosion control treatment for Portland's water were considered: limited, moderate, and LCR-defined optimal. As described in Section 2.6, these treatment levels involve pH adjustment to 7.5, 8.5, and 9.5, respectively, with predicted percent reductions in water lead levels of 40%, 60%, and 70%, respectively, as compared with water lead levels before treatment.

## 4.2 Results

Tables 4-2 through 4-5 and Figure 4-2 show the predicted contributions of water lead to blood lead levels for infants, children, and adults.

**Table 4-2**
**Individual-Based Modeling: INFANTS (0-6 months)**
**Estimated Contribution of Water Lead to Blood Lead Levels [1]**

| pH 6.5-7.0 | no corrosion control treatment | | pH 7.0-7.5 | limited treatment | pH 8.0-8.5 | moderate treatment | pH 9.0-9.5 | "optimal" treatment |
|---|---|---|---|---|---|---|---|---|
| | | | 40% reduction in water lead | | 60% reduction in water lead | | 70% reduction in water lead | |
| Assuming consumption of water with lead level (ug/L) of: | Relevance of water lead level: | Water lead contribution to blood lead level (ug/dL) | Assuming consumption of water with lead level (ug/L) of: | Water lead contribution to blood lead level (ug/dL) | Assuming consumption of water with lead level (ug/L) of: | Water lead contribution to blood lead level (ug/dL) | Assuming consumption of water with lead level (ug/L) of: | Water lead contribution to blood lead level (ug/dL) |
| 1 | < 1 ug/L = 50th %tile lead level, running water, Portland homes [2] | 0.3 | 0.6 | 0.2 | 0.4 | 0.1 | 0.3 | 0.1 |
| 3 | 90th %tile lead level, running water, Portland homes [2] | 0.8 | 1.8 | 0.5 | 1.2 | 0.3 | 0.9 | 0.2 |
| 4 | 50th %tile lead level, standing water, Portland homes [2] | 1.0 | 2.4 | 0.7 | 1.6 | 0.6 | 1.2 | 0.3 |
| 5 | FDA's lead limit in bottled water | 1.3 | No treatment after bottling | No treatment after bottling | No treatment after bottling | No treatment after bottling | No treatment after bottling | No treatment after bottling |
| 10 | 50th %tile lead level, standing water, Portland Tier 1 homes [3] | 2.6 | 6.0 | 1.6 | 4.0 | 1.0 | 3.0 | 0.8 |
| 14 | 90th %tile lead level, standing water, Portland homes [2] | 3.6 | 8.4 | 2.2 | 5.6 | 1.5 | 4.2 | 1.1 |
| 49 | 90th %tile lead level, standing water, Portland Tier 1 homes [3] | 5.3 | 29 | 4.5 | 20 | 4.1 | 15 | 3.8 |

(1) Using EPA-preferred (USEPA, 1991) absorption coefficients from Lacey and others (1985); see Table 4-1.
(2) Portland customer requests for free lead-in water analysis; see Table 2-2.
(3) Portland LCR initial tap monitoring data (pooled data from two monitoring rounds conducted in 1992); see Table 2-1.

**Table 4-3**
**Individual-Based Modeling: CHILDREN (> 6 months to < 6 years)**

**Estimated Contribution of Water Lead to Blood Lead Levels [1]**

| pH 6.5-7.0 | no corrosion control treatment | | pH 7.0-7.5 | limited treatment | pH 8.0-8.5 | moderate treatment | pH 9.0-9.5 | "optimal" treatment |
|---|---|---|---|---|---|---|---|---|
| | | | 40% reduction in water lead | | 60% reduction in water lead | | 70% reduction in water lead | |
| Assuming consumption of water with lead level (ug/L) of: | Relevance of water lead level: | Water lead contribution to blood lead level (ug/dL) | Assuming consumption of water with lead level (ug/L) of: | Water lead contribution to blood lead level (ug/dL) | Assuming consumption of water with lead level (ug/L) of: | Water lead contribution to blood lead level (ug/dL) | Assuming consumption of water with lead level (ug/L) of: | Water lead contribution to blood lead level (ug/dL) |
| 1 | < 1 ug/L = 50th %tile lead level, running water, Portland homes [2] | 0.1 | 0.6 | <0.1 | 0.4 | <0.1 | 0.3 | < 0.1 |
| 3 | 90th %tile lead level, running water, Portland homes [2] | 0.4 | 1.8 | 0.2 | 1.2 | 0.1 | 0.9 | 0.1 |
| 4 | 50th %tile lead level, standing water, Portland homes [2] | 0.5 | 2.4 | 0.3 | 1.6 | 0.2 | 1.2 | 0.1 |
| 5 | FDA's limit in bottled water | 0.6 | No treatment after bottling | No treatment after bottling | No treatment after bottling | No treatment after bottling | No treatment after bottling | No treatment after bottling |
| 10 | 50th %tile lead level, standing water, Portland Tier 1 homes [3] | 1.2 | 6.0 | 0.7 | 4.0 | 0.5 | 3.0 | 0.4 |
| 14 | 90th %tile lead level, standing water, Portland homes [2] | 1.7 | 8.4 | 1.0 | 5.6 | 0.7 | 4.2 | 0.5 |
| 49 | 90th %tile lead level, standing water, Portland Tier 1 homes [3] | 3.8 | 29 | 2.6 | 20 | 2.1 | 15 | 1.8 |

(1) Using EPA-preferred (USEPA, 1991) absorption coefficients from Lacey and others (1985); see 4-1.
(2) Portland customer requests for free lead-in water analysis; see Table 2-2.
(3) Portland LCR initial tap monitoring data (pooled data from two monitoring rounds conducted in 1992); see Table 2-1.

**Table 4-4**
**Individual-Based Modeling: ADULTS**
**Estimated Contribution of Water Lead to Blood Lead Levels [1]**

| pH 6.5-7.0 | no corrosion control treatment | | pH 7.0-7.5 | limited treatment | pH 8.0-8.5 | moderate treatment | pH 9.0-9.5 | "optimal" treatment |
|---|---|---|---|---|---|---|---|---|
| | | | 40% reduction in water lead | | 60% reduction in water lead | | 70% reduction in water lead | |
| Assuming consumption of water with lead level (ug/L) of: | Relevance of water lead level: | Water lead contribution to blood lead level (ug/dL) | Assuming consumption of water with lead level (ug/L) of: | Water lead contribution to blood lead level (ug/dL) | Assuming consumption of water with lead level (ug/L) of: | Water lead contribution to blood lead level (ug/dL) | Assuming consumption of water with lead level (ug/L) of: | Water lead contribution to blood lead level (ug/dL) |
| 1 | < 1 ug/L = 50th %tile lead level, running water, Portland homes [2] | <0.1 | 0.6 | <0.1 | 0.4 | <0.1 | 0.3 | < 0.1 |
| 3 | 90th %tile lead level, running water, Portland homes [2] | 0.2 | 1.8 | 0.1 | 1.2 | 0.1 | 0.9 | 0.1 |
| 4 | 50th %tile lead level, standing water, Portland homes [2] | 0.2 | 2.4 | 0.1 | 1.6 | 0.1 | 1.2 | 0.1 |
| 5 | FDA's lead limit in bottled water | 0.3 | No treatment after bottling | No treatment after bottling | No treatment after bottling | No treatment after bottling | No treatment after bottling | No treatment after bottling |
| 10 | 50th %tile lead level, standing water, Portland Tier 1 homes [3] | 0.6 | 6.0 | 0.4 | 4.0 | 0.2 | 3.0 | 0.2 |
| 14 | 90th %tile lead level, standing water, Portland homes [2] | 0.8 | 8.4 | 0.5 | 5.6 | 0.3 | 4.2 | 0.3 |
| 49 | 90th %tile lead level, standing water, Portland Tier 1 homes [3] | 2.9 | 29 | 1.7 | 20 | 1.2 | 15 | 0.9 |

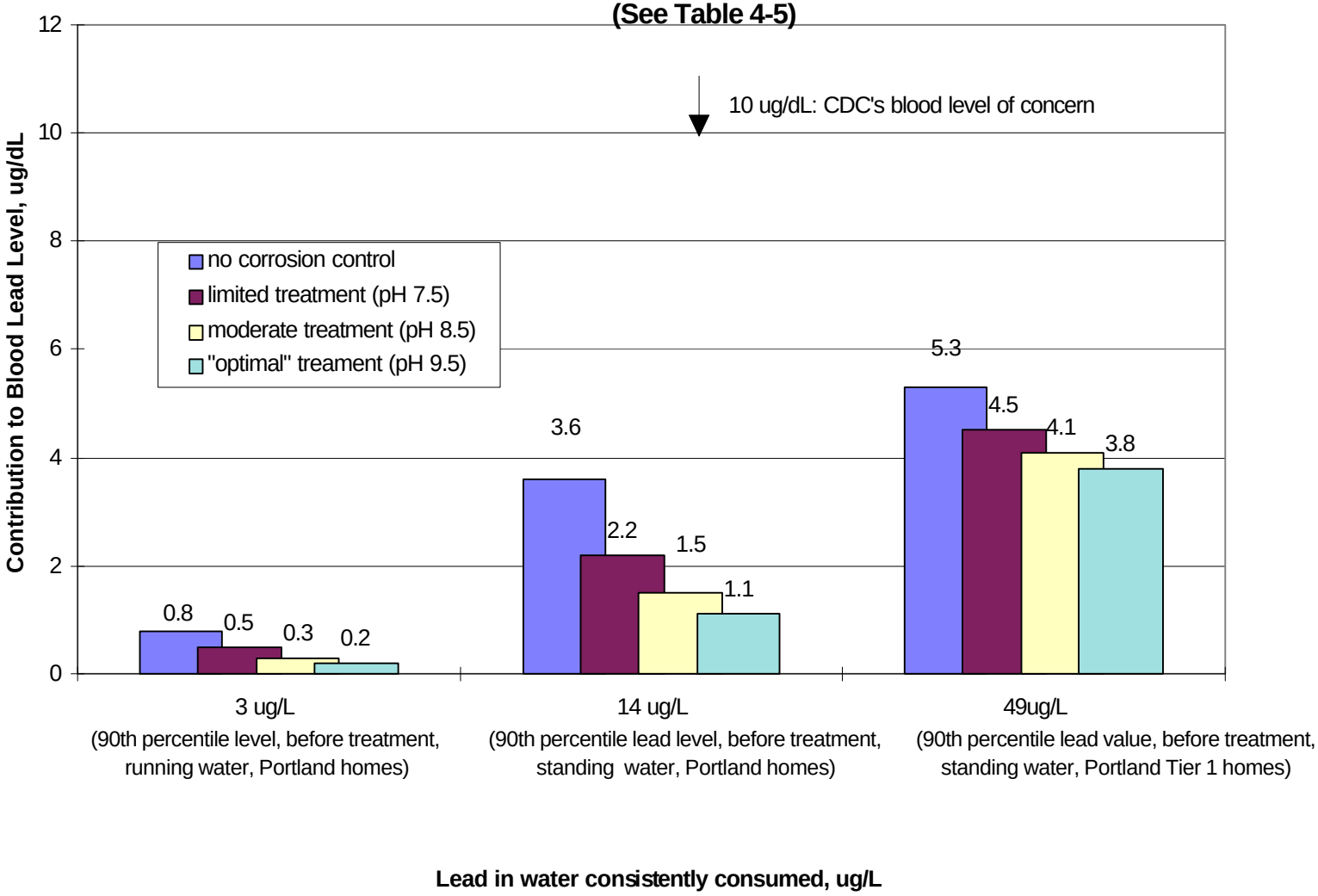
(1) Using EPA-preferred (USEPA, 1991) absorption coefficients from Lacey and others (1985); see Table 4-1.
(2) Portland customer requests for free lead-in water analysis; see Table 2-2.
(3) Portland LCR initial tap monitoring data (pooled data from two monitoring rounds conducted in 1992); see Table 2-1.

**Table 4-5**
**Summary of Results of Individual-Based Modeling:**
**Estimated Contribution of Water Lead to Blood Lead Levels for**
**INFANTS, CHILDREN, and ADULTS**
**(Results summarized from Tables 4-2, 4-3, and 4-4)**

| Assuming consumption of water with lead level (ug/L) of: | Relevance of water lead level: | Water lead contribution to blood lead level (ug/dL) | Water lead contribution to blood lead level (ug/dL) ---------- with 40% reduction in water lead level with limited corrosion control treatment ( pH 7.5) | Water lead contribution to blood lead level (ug/dL) ---------- with 60% reduction in water lead level with moderate corrosion control treatment (pH 8.5) | Water lead contribution to blood lead level (ug/dL) ---------- with 70% reduction in water lead level with LCR-defined optimal corrosion control treatment ( pH 9.5) | Difference in water lead contribution to blood lead level (ug/dL) between pH 7.5 and pH 9.5 |
|---|---|---|---|---|---|---|
| 1 | <1 ug/L= 50th %tile lead level, running water, Portland homes | Infant: 0.3<br>Child: 0.1<br>Adult: <0.1 | 0.2<br><0.1<br><0.1 | 0.1<br><0.1<br><0.1 | 0.1<br><0.1<br><0.1 | 0.1<br><0.1<br><0.1 |
| 3 | 90th %tile lead level, running water, Portland homes (1) | Infant: 0.8<br>Child: 0.4<br>Adult: 0.2 | 0.5<br>0.2<br>0.1 | 0.3<br>0.1<br>0.1 | 0.2<br>0.1<br>0.1 | 0.3<br>0.1<br>0.0 |
| 5 | FDA's lead limit in bottled water | Infant: 1.3<br>Child: 0.6<br>Adult: 0.3 | No treatment after bottling | No treatment after bottling | No treatment after bottling | No treatment after bottling |
| 14 | 90th %tile lead level, standing water, Portland homes (1) | Infant 3.6<br>Child: 1.7<br>Adult: 0.8 | 2.2<br>1.0<br>0.5 | 1.5<br>0.7<br>0.3 | 1.1<br>0.5<br>0.3 | 1.1<br>0.5<br>0.2 |
| 49 | 90th %tile lead level, standing water, Portland Tier 1 homes (2) | Infant 5.3<br>Child: 3.8<br>Adult: 2.9 | 4.5<br>2.6<br>1.7 | 4.1<br>2.1<br>1.2 | 3.8<br>1.8<br>0.9 | 0.7<br>0.8<br>0.8 |

(1) Portland customer requests for free lead-in water analysis; see Table 2-2.
(2) Portland LCR initial tap monitoring data (pooled data from two monitoring rounds conducted in 1992); see Table 2-1.

**Figure 4-2**
**Estimated Contribution of Water Lead to Blood Lead Levels for INFANTS in Portland**
**(See Table 4-5)**



In Portland, the median lead level in running samples is estimated at < 1 ug/L (below the detection limit). An estimated 95% of the running water tap samples and 70% of the standing water tap samples in Portland meet the FDA limit of 5 ug/L for lead in bottled water (Table 2-2). Regular consumption of water containing lead at the 5 ug/L level is estimated to contribute about 1.3, 0.6, and 0.3 ug/dL to an infant's, child's, and adult's blood lead level, respectively, toward the 10 ug/dL blood lead level of concern. (Table 4-5)

In order for water to significantly contribute to an individual's blood lead level, that individual's water consumption would likely have to consist solely of standing water with elevated lead levels. Consider the exposure scenario of an infant consistently consuming standing water with 49 ug/L of lead (the 90th percentile standing lead level in Portland's Tier 1 homes in 1992). A blood lead level contribution of 5.3 ug/dL from water is predicted, representing a contribution of about 50% of the 10 ug/dL level of concern. (Table 4-5 and Figure 4-2)

In some homes where significantly elevated levels of lead in standing water occur, if an individual's water consumption consists solely of standing water, it is predicted that even LCR-defined optimal corrosion control treatment would not prevent lead in water from substantially contributing to an individual's total lead exposure. Consider again the exposure scenario of an infant regularly consuming standing water with 49 ug/L of lead (the 90th percentile standing lead level in Portland's Tier 1 homes in 1992) and experiencing a blood lead level contribution of 5.3 ug/dL. LCR-defined optimal corrosion control treatment is predicted to reduce the water lead level by 70% to 15 ug/L, which is predicted to result in a still substantial blood lead level contribution of 3.8 ug/dL, about 40% of the 10 ug/dL blood lead level of concern (Table 4-5 and Figure 4-2). So the difference between the no corrosion treatment and optimal corrosion treatment options is only an estimated 10% reduction in blood lead levels.

In summary, the differences in reductions of individual blood lead level contributions from water predicted as a result of a limited level of corrosion control treatment (pH adjustment to 7.5) as compared to LCR-defined optimal treatment (pH adjustment to 9.5) are typically less than 1 ug/dL (Table 4-5).

# Section 5 Population-based Modeling

## 5.1 Description of the Model

Population-based modeling was used to compare the existing distribution of blood lead levels in Portland's population before corrosion control treatment with predicted distributions after implementation of various levels of corrosion control treatment.

Various exposure scenarios were modeled, each consisting of three components: a corrosion control treatment level of interest, a population group of interest, and an assumption regarding the consumption of running and/or standing water. Thirty-six exposure scenarios were considered, involving all the combinations of:

- 3 levels of corrosion control treatment (limited, moderate, and LCR-defined optimal);
- 4 population groups (infants, children, adults, and total population)
- 3 assumptions for running and/or standing water consumption (only running water, only standing water, and a mix).

The three components, the options under each component, and associated input to the model are summarized in Table 5-1.

**Table 5-1**
**Population-based Exposure Scenarios and Associated Model Inputs**

| Components of an Exposure Scenario | Related Model Input | Options for Each Component | Model Input |
|---|---|---|---|
| Assumption regarding consumption of running and/or standing water | Appropriate distribution of water lead levels | ❑ Consistent consumption of only running water | ***1A***: Distribution of lead levels in running water samples from Portland's Customer Request data base - before corrosion control treatment (Table 2-2) |
| | | ❑ Consistent consumption of only standing water | ***1B***: Distribution of lead levels in standing water samples from Portland's Customer Request data base - before corrosion control treatment (Table 2-2) |
| | | ❑ Consistent consumption of a mix of 75% running and 25% standing water | ***1C***: Weighted combination of the water lead distributions described above |
| Corrosion control treatment level | Predicted extent of water lead reductions associated with treatment | ❑ Limited treatment (pH adjustment up to 7.5) | ***2A***: 40% reduction in water lead levels compared to no treatment (Table 2-4) |
| | | ❑ Moderate treatment (pH adjustment up to 8.5) | ***2B***: 60% reduction in water lead levels compared to no treatment (Table 2-4) |
| | | ❑ "Optimal" treatment (pH adjustment up to 9.5) | ***2C***: 70% reduction in water lead levels compared to no treatment (Table 2- 4) |
| Population group | Appropriate coefficients relating water lead levels to contributions to blood lead levels | ❑ Infants in Portland (0-6 mos) | ***3A***: EPA-preferred coefficients (Table 4-1) |
| | | ❑ Children in Portland (> 6 mos to < 6 years) | ***3B***: EPA-preferred coefficients (Table 4-1) |
| | | ❑ Adults in Portland | ***3C***: EPA-preferred coefficients (Table 4-1) |
| | | ❑ Total population | No model input * |
| | Appropriate distribution of blood lead levels | ❑ Infants in Portland (0-6 mos) | ***4A***: Distribution of infant blood lead levels in Multnomah County, OCLPPP data - before corrosion control treatment (Table 3-2) |
| | | ❑ Children in Portland (> 6 mos. to < 6 years) | ***4B***: Distribution of child blood lead levels in Multnomah County, OCLPPP data - before corrosion control treatment (Table 3-2) |
| | | ❑ Adults in Portland | ***4C***: Distribution of adult blood lead levels in U.S., NHANES III data (Table 3-1) |
| | | ❑ Total population | No model input * |

* Predicted blood lead distribution after corrosion control treatment (model output) for the total population was calculated as a weighted composite of infant, child, and adult distributions. Weighting factors were determined based the percentages of infants, children, and adults in Portland (0.65%, 8.87%, and 90.48% , respectively) calculated from 1990 U.S. Census data.

Assumption 1A in Table 5-1 is that the entire population's water consumption consists solely of running water (water that has had little or no standing contact time with premise plumbing materials); this is a " best case" (least lead) assumption. Assumption 1B in Table 5-1 is that the entire population's water consumption consists solely of standing

water (water that has stood in contact with premise plumbing materials for at least 6 hours); this represents an unrealistic "worst-case" assumption. Assumption 1C in Table 5-1 is that the entire population's water consumption consists of 75% running water and 25% standing water; this is intended to be a conservative assumption that recognizes that both running and standing water are consumed.

For each of the 36 exposure scenarios described above, a predicted blood lead distribution resulting from corrosion control treatment was calculated as follows:

- For each of 100 water lead values equivalent to the $1^{st}$,$2^{nd}$,$3^{rd}$ , ..., $100^{th}$ percentile values from the *appropriate water lead distribution (1A, 1B, or 1C in Table 5-1):*
    - a predicted reduced water lead value was calculated using the *appropriate water lead reduction factor (2A, 2B, or 2C in Table 5-1);*
    - a corresponding predicted value of blood lead level reduction was calculated using the *appropriate coefficient(s) relating water lead levels to contributions to blood lead levels (3A, 3B, or 3C in Table 5-1);*
    - a predicted, reduced new blood lead distribution was calculated by subtracting the predicted value of blood lead level reduction calculated above from each of 100 blood lead values equivalent to the values $1^{st}$,$2^{nd}$,$3^{rd}$ , ..., $100^{th}$ percentile values from the *appropriate blood lead distribution (4A, 4B, or 4C in Table 5-1)*.
- This results in 100 new blood lead distributions, associated with $1^{st}$,$2^{nd}$,$3^{rd}$ , ..., $100^{th}$ percentile values of the water lead distribution, and each having probability of occurrence of p=0.1. These distributions were integrated to calculate the predicted reduced blood lead distribution for the population.

## 5.2 Results

For each of the 36 exposure scenarios described above, a predicted "after treatment" blood lead distribution was calculated. Each predicted blood lead value (the $1^{st}$,$2^{nd}$,$3^{rd}$ , ..., $100^{th}$ percentile value) in the "after treatment" distribution is a constant amount lower than the corresponding "before treatment" value. The constant value of the blood lead reduction varies depending on the exposure scenario.

In Table 5-2, two measures of comparison are tabulated for each of the exposure scenarios:

- the predicted constant value of blood lead reduction that is applied to the "before treatment" distribution to obtain the "after treatment" distribution; and
- the predicted decrease in the percent of population with blood lead levels at or above the 10 ug/dL blood lead level of concern.

Figures 5-1A, 5-1B, 5-1C, and 5-1D present the "before treatment" and predicted "after treatment" blood lead distributions for infants, children, adults, and the total population, respectively.

**Table 5-2**
**Summary of Results for Population-Based Modeling**

| | **Assuming consistent consumption of this water by all population members** | **Predicted reduction in blood lead level (ug/dL) from the "before treatment" blood lead distribution** | | | **Predicted percentage of population with elevated blood lead levels (≥10 ug/dL) as compared to "before treatment" percentages of :**<br><br>**5.9% for infants**<br>**5.9% for children**<br>**4.6% for adults**<br>**4.6 % for total population** | | |
|---|---|---|---|---|---|---|---|
| | | pH 7.5 | pH 8.5 | pH 9.5 | pH 7.5 | pH 8.5 | pH 9.5 |
| Infants | 100% running water | 0.2 | 0.3 | 0.3 | 5.9% | 5.8% | 5.8% |
| | 75% running/ 25% standing water | 0.3 | 0.4 | 0.5 | 5.8% | 5.8% | 5.8% |
| | 100% standing water | 0.5 | 0.8 | 0.9 | 5.8% | 5.8% | 5.8% |
| Children | 100% running water | 0.1 | 0.1 | 0.1 | 5.9% | 5.9% | 5.9% |
| | 75% running/ 25% standing water | 0.1 | 0.2 | 0.2 | 5.9% | 5.8% | 5.8% |
| | 100% standing water | 0.2 | 0.3 | 0.4 | 5.8% | 5.8% | 5.8% |
| Adults | 100% running water | <0.1 | <0.1 | <0.1 | 4.6% | 4.6% | 4.6% |
| | 75% running/ 25% standing water | <0.1 | <0.1 | 0.1 | 4.6% | 4.6% | 4.6% |
| | 100% standing water | 0.1 | 0.2 | 0.2 | 4.6% | 4.6% | 4.6% |
| Total population | 100% running water | <0.1 | <0.1 | <0.1 | 4.6% | 4.6% | 4.6% |
| | 75% running/ 25% standing water | <0.1 | 0.1 | 0.1 | 4.6% | 4.6% | 4.6% |
| | 100% standing water | 0.1 | 0.2 | 0.2 | 4.6% | 4.6% | 4.6% |

**Figure 5-1A**
**Predicted Blood Level Distributions After Corrosion Control Treatment**
**Infants in Portland**

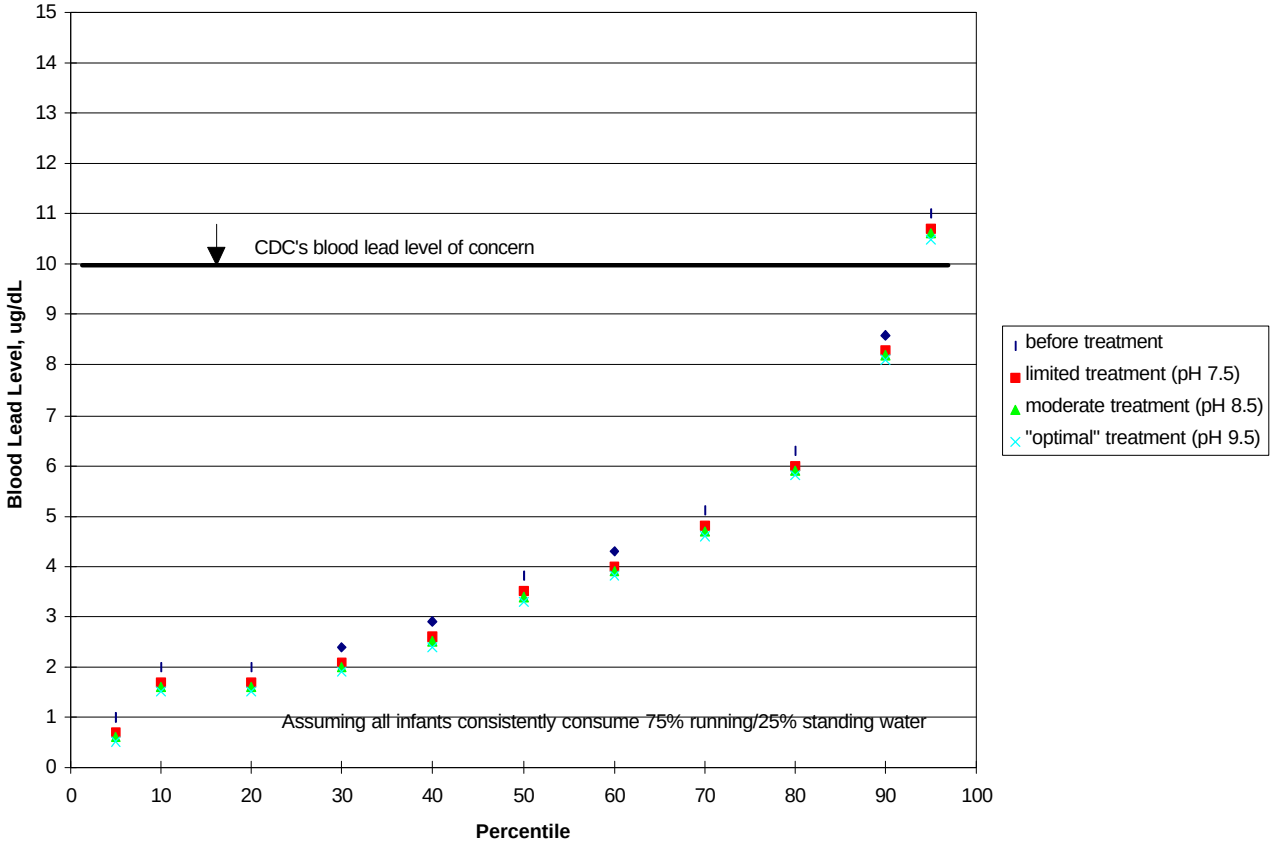


**Figure 5-1B**
**Predicted Blood Lead Level Distributions After Corrosion Control Treatment Children in Portland**

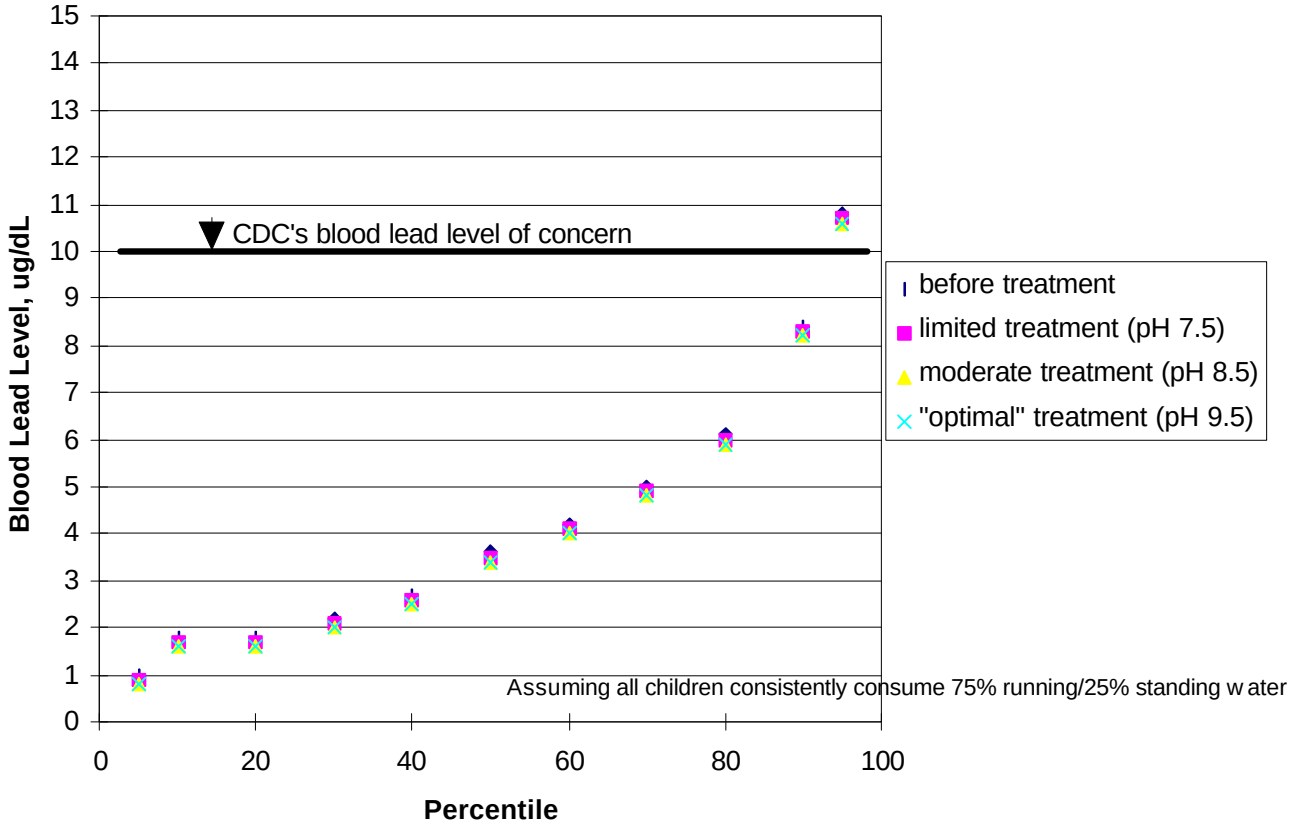


**Figure 5-1C**
**Predicted Blood Level Distrubutions After Corrosion Control Treatment**
**Adults in Portland**

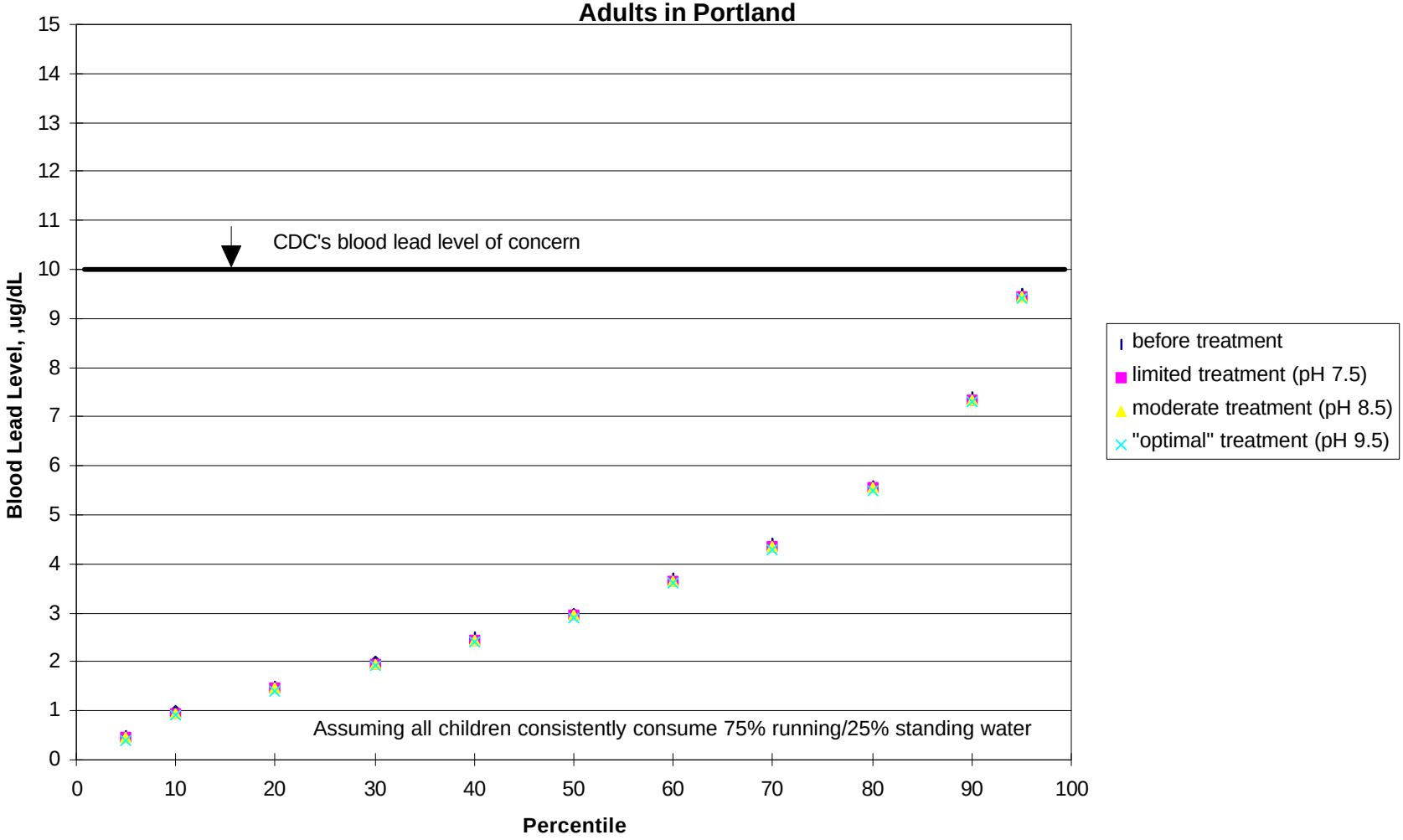
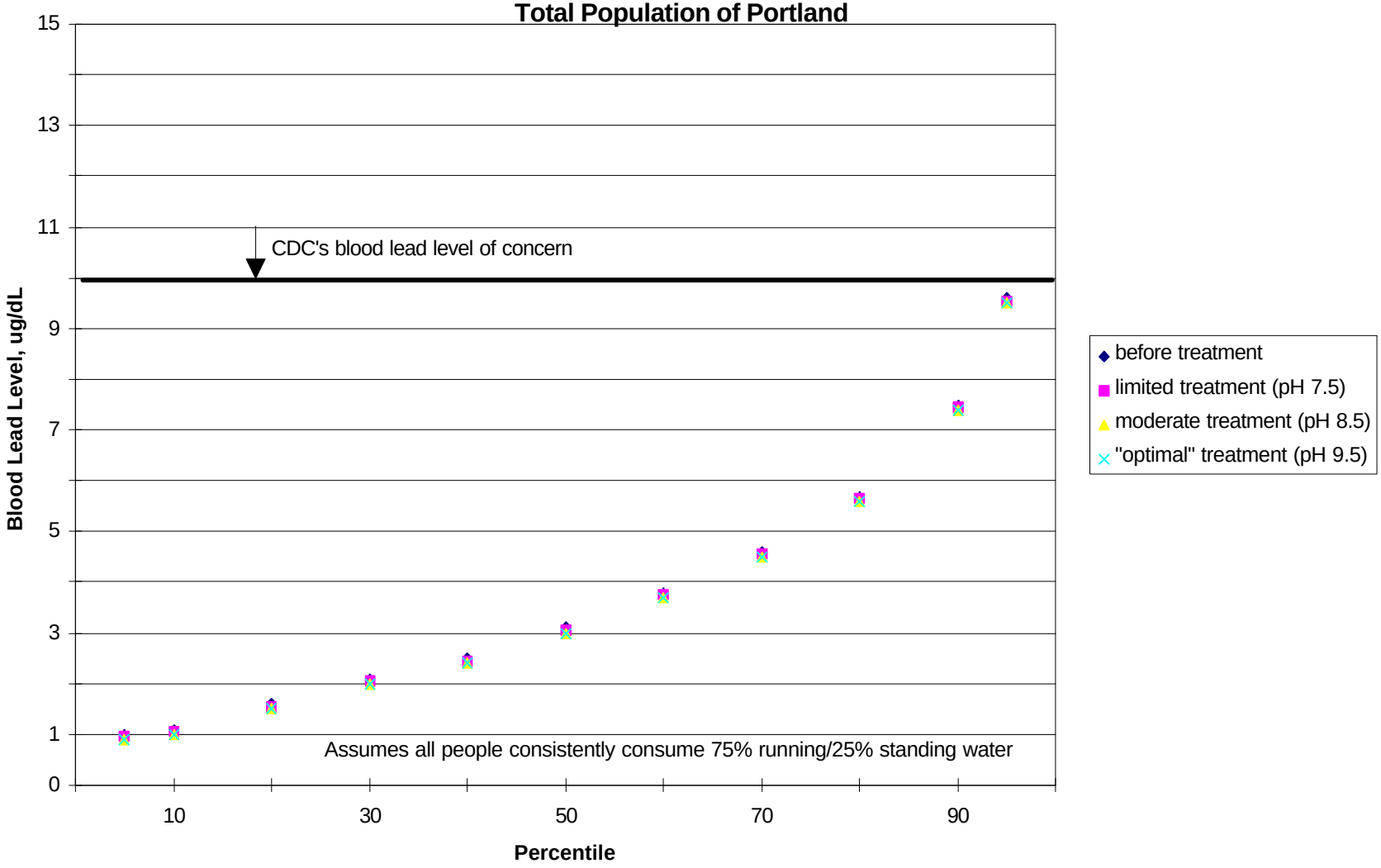


**Figure 5-1D**
**Predicted Blood Level Distributions After Corrosion Control Treatment**
**Total Population of Portland**

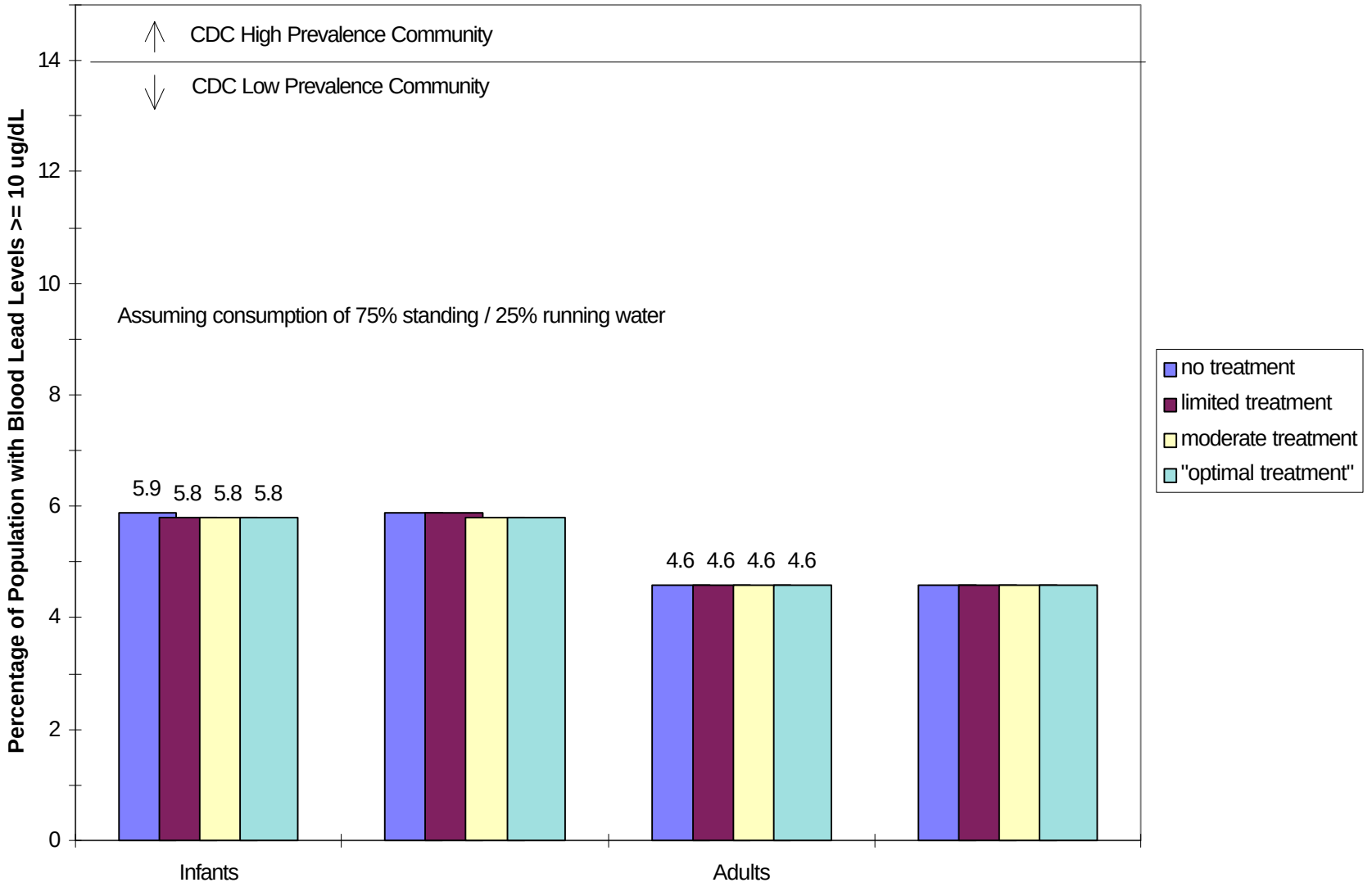


As an example of how the data in Table 5-2 and Figures 5-1A, 5-1B, 5-1C, and 5-1D are interpreted, consider the scenario of infants consistently consuming 75% running / 25% standing water. The "before treatment" median, 90th, and 95th percentile blood lead levels for this group are estimated at 3.8, 8.6, and 11 ug/dL respectively (Figure 5-1A). With optimal corrosion control treatment, each of these blood lead percentiles are predicted to decrease by 0.5 ug/dL (Table 5-2) to 3.3, 8.1, and 10.5 ug/dL, respectively (Figure 5-1A).

Table 5-2 shows that the amount by which the "before treatment" and predicted "after treatment" blood lead distributions differ is < 1 ug/dL for all scenarios. For example, for the conservative assumption that water consumption consists of 75% running /25% standing water, the "before treatment" blood lead distributions for all population groups are shifted downwards by <= 0.5 ug/dL to obtain the "after treatment" blood lead distributions. The amount by which the predicted blood lead distributions for "limited treatment" and "optimal treatment" scenarios differ is typically <= 0.2 ug/dL.

The CDC (1995) proposed a definition of low-prevalence communities as those in which 14% or less of children have blood lead levels >= 10 ug/dL. As indicated in Table 5-2, the percentage of Portland's population with elevated (>= 10 ug/dL) blood lead levels before corrosion control treatment is less than 6% for all population age groups considered. Thus, Portland would be considered a low-prevalence community under this definition. The data in Table 5-2 and Figure 5-2 show that the predicted percentage of the population with elevated blood lead levels would essentially remain unchanged as a result of corrosion control treatment.

**Figure 5-2**
**Percent of Portland's Population with Elevated Blood Lead Levels for Various Levels of**



# Section 6 - Conclusions

## 6.1 Health Effects of Lead Exposure

Lead in the environment is associated with a variety of adverse health effects. Lead exposure across a broad range of blood lead levels has been associated with a spectrum of pathophysiological effects, including interference with heme synthesis, anemia, kidney damage, impaired reproductive function, interference with vitamin D metabolism, impaired cognitive performance, delayed neurological and physical development, and elevations in blood pressure (USEPA, 1991). Lead is most hazardous to children under the age of six, whose still developing nervous systems are particularly vulnerable to lead and whose normal activities expose them to lead-contaminated dust and soil.

The Centers for Disease Control (CDC) (1991) indicates that epidemiologic studies have identified harmful effects of lead in children with blood lead levels as low as 10 ug/dL. These blood lead levels do not cause distinctive symptoms, but have been associated with small decrements on intelligence tests and some indications of delayed neurobehavioral development. Some studies have suggested harmful effects at even lower levels, but the body of information accumulated so far is not adequate for effects below about 10 ug/dL to be evaluated definitively.

The CDC recommends the following multi-tier approach to follow-up, with the goal of all lead poisoning prevention activities being to reduce children's blood lead levels below 10 ug/dL:

- Community-wide interventions: If many children in the community have blood lead levels at or above 10 ug/dL, community-wide efforts to *prevent* exposure should be considered. The CDC (1995) proposed a definition of high-prevalence communities as those in which 14% or more of children have blood lead levels >= 10 ug/dL.

- Interventions for individual children should begin at blood lead levels of 15 ug/dL.

    - For 10-14 ug/dL: Several reasons are cited for not recommending interventions for children with blood lead levels in this range, including: inaccurate and imprecise laboratory measurements, lack of effective interventions identified for blood lead levels in this range, and diversion of resources away from follow-up of children with higher blood lead levels.

    - For 15-19 ug/dL: Children are at risk for subtle adverse effects including decreases in IQ of up to several IQ points. Educational and nutritional interventions and follow-up testing are recommended; if levels persist above 15 ug/dL, environmental investigation and remediation is

recommended.

- For > 19 ug/dL: Medical evaluation, and environmental evaluation and remediation are recommended; levels >44 ug/dL require urgent medical follow-up.

- When resources are limited, the highest priority for interventions should be the children with the highest blood lead levels.

- When possible, abatement should be conducted for primary prevention of lead poisoning.

The EPA (1995) states that interventions should not be performed merely to reduce or eliminate environmental lead levels; the aim is always to positively impact the health of children or adults. Intervention to reduce lead exposures should be targeted at those exposure pathways that have the greatest impact on the health of the child by reducing his or her body-lead burden. An intervention can reduce a child's lead exposure no more than that consistent with the source of exposure targeted. Potentially, an intervention can be successful in reducing a particular environmental lead exposure and yet produce no positive impact in a child only marginally exposed to the abated lead hazard.

## 6.2 Conclusions from this Study

- <u>The prevalence of elevated blood lead levels in Portland is low.</u>

  The most recent National Health and Nutrition Survey (NHANES III) (Brody and others, 1994) indicates that blood lead levels across the nation have decreased dramatically from the levels that were known when the Lead and Copper Rule was promulgated in 1991. Existing blood lead levels of children in Multnomah County (the Oregon county in which Portland is located) are consistent with the blood lead levels of children reported in the NHANES III study (Section 3.5).

  The CDC (1995) proposed a definition of low-prevalence communities as those in which 13% or less of children have blood lead levels >= 10 ug/dL. Because an estimated 6% of children in Portland have such levels (Table 3-4), Portland would be considered a low-prevalence community under this definition.

- <u>Lead-based paint is the most commonly identified source of elevated blood lead levels in Multnomah County</u>.

  The Multnomah County Health Department (MCHD) has conducted over 120 follow-up investigations of elevated blood lead levels in Multnomah County and the Portland metropolitan area since 1993. Analysis of the results of these

investigations indicates that lead-based paint is the most likely source of exposure for 70% of the EBLLs of at least 15 ug/dL, and for 80% of the EBLLs of at least 20 ug/dL (OHD, 1997). These observations are consistent with the CDC's statement that lead-based paint is the most common high-dose source of lead exposure for children (CDC, 1991).

- Water is not a major route of lead exposure in Portland.

  Based on analysis of best available data, the median lead level in running samples is estimated at < 1 ug/L (below the detection limit). The FDA has set a limit of 5 ug/L limit on lead in bottled water. It is estimated that about 95% of the running tap water samples and about 70% of the standing tap water samples in Portland meet the FDA standard (Table 2-2).

  However, for a set of unlikely exposure conditions, it is possible that water could provide a significant contribution to an individual's blood lead level. In order for water to significantly contribute to an individual's blood lead level, that individual's water consumption would likely have to consist solely of standing water with elevated lead levels.

  For example, consider the highly unlikely exposure scenario of an individual infant consistently consuming standing water with 49 ug/L of lead (the 90th percentile standing lead level in Portland's Tier 1 homes in 1992). A blood lead level contribution of about 5 ug/dL from water is predicted for this infant (Section 4.2).

  The real questions become: To what extent would corrosion control treatment be expected to reduce contributions to blood lead levels from water, and what health benefits, if any, may result?

- Optimal corrosion control treatment would provide only minimal reduction in the contributions from water to an individual's blood lead level.

  Consider again the highly unlikely but possible exposure scenario of an individual infant consistently consuming standing water with 49 ug/L of lead (the 90th percentile standing lead level in Portland's Tier 1 homes in 1992). Optimal corrosion control treatment would be expected to reduce a "before treatment" water lead level of 49 ug/L by about 70% to 15 ug/L; this would correspond to a minimal decrease in blood lead contribution for an infant from about 5 to 4 ug/dL (Section 4.2). In such cases, other interventions such as tap flushing or replacement of lead-bearing materials in premise plumbing would be needed.

- Even with optimal corrosion control treatment, the predicted percentage of the population with elevated blood lead levels would essentially remain unchanged.

  The population-based model predicts that the amount by which the "before

treatment" and predicted "after treatment" blood lead distributions in Portland differ would be < 1 ug/dL. For example, consider the conservative exposure scenario in which Portland infants consistently consume a mix of running (75%) and standing (25%) water. The "before treatment" median, 90th, and 95th percentile blood lead levels for this group are estimated at 3.8, 8.6, and 11.0 ug/dL, respectively. With corrosion control treatment involving pH adjustment to 7.5-9.5, each of these percentiles is predicted to decrease by only 0.3-0.5 ug/dL (Table 5-2).

- There is very little difference between the blood lead level reductions that can be expected with limited corrosion control treatment (pH adjustment to 7.5) as compared to LCR-defined optimal corrosion control treatment (pH adjustment to 9.5).

  Population-based modeling predicts that the amount by which the predicted blood lead distributions for "limited treatment" and "optimal treatment" scenarios differ is typically <= 0.2 ug/dL (Table 5-2).

  Individual-based modeling predicts that the differences in reductions of individual blood lead level contributions from water with a limited level of corrosion control treatment (pH adjustment to 7.5) as compared to LCR-defined optimal treatment (pH adjustment to 9.5) are typically less than 1 ug/dL (Table 4-5).

- While corrosion control treatment of Portland's Bull Run water supply would be expected to reduce lead in water by 40-70% as compared to "no treatment" levels (Table 2-4), the type and extent of any resulting health benefits are much less certain.

  For children with blood lead levels already below the 10 ug/dL level of concern, there is currently no conclusive evidence that reducing blood lead levels further, especially by the levels predicted with corrosion control treatment, would provide any health benefits. Again, Portland may be considered a community in which there is a low-prevalence of elevated blood levels. Even with optimal corrosion control treatment, the predicted percentage of the population with elevated blood lead levels would essentially remain unchanged.

  For children with blood lead levels that are well above the 10 ug/dL level of concern, corrosion control is not likely to reduce these levels to below 10 ug/dL.

- Interventions to reduce lead exposures should be targeted at those exposure pathways most significantly contributing to a child's total exposure.

  Since lead-based paint is the most commonly identified source of elevated blood lead levels in Portland, interventions should be focused on this source.

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